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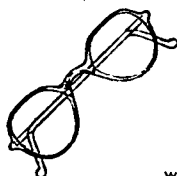
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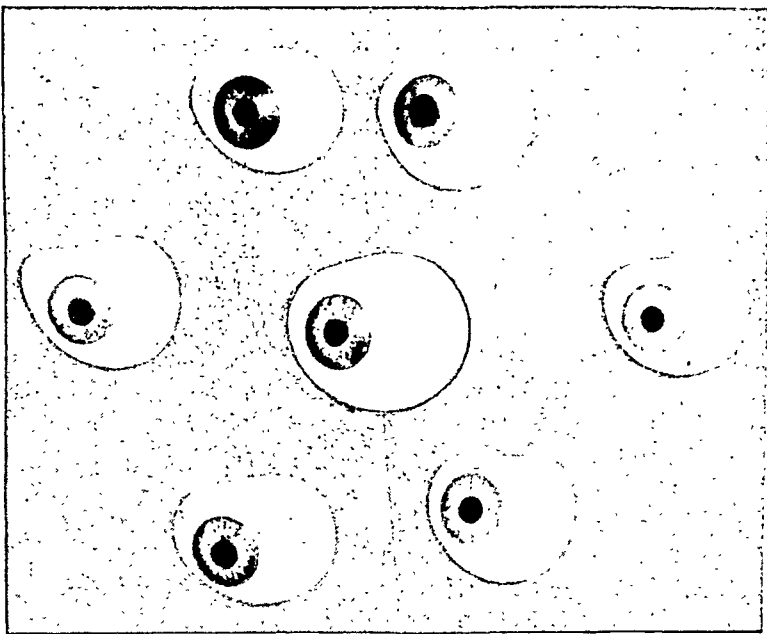
THE BRITISH JOURNAL OF OPHTHALMOLOGY LTD.

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ARTIFICIAL EYES IN PLASTIC

Artificial Eyes until the beginning of the war were invariably made of glass, and their production was entirely in the hands of a few very skilled craftsmen in this Country and on the Continent. Up to August, 1939, Theodore Hamblin, Ltd., employed Mr. Paul Asprien, of Vienna, at 15, Wigmore Street, and at their various provincial branches, where he made artificial eyes in glass while the patient waited. With the outbreak of war, these visits had to cease and steps were taken to develop the manufacture of artificial eyes in plastic material. The many difficulties of producing eyes in this material have been overcome, and they are now made throughout in plastic, no paper or glass being incorporated.



Eyes made in plastic have many advantages over those made in glass. They are life-like in appearance, comfortable in wear, are not affected by the secretions of the orbit, and above all, they are unbreakable.

Difficult shapes necessitated by war injuries, burns, etc., or thin shells to fit over shrunken or deformed globes, almost impossible to produce in glass, are quite possible in plastic.

Patients may be sent in to 15, Wigmore Street or to most of the provincial branches, where stocks of ready-made eyes are available from which selections may be made and fitted. Specially made eyes for more difficult orbits can be made with little delay. For these a carefully made mould of the orbit is first taken, and a special iris is produced in plastic. In such cases a second visit after the eye has been made is necessary for fitting.

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THE BRITISH JOURNAL OF OPHTHALMOLOGY

JANUARY, 1947

COMMUNICATIONS

FUTURE POLICY

With this issue of the Journal we are happy to state that it is now possible to use a much better quality paper than war conditions imposed and we hope that this improvement in appearance will be welcomed by our subscribers.

Certain changes in policy have been considered by the Management and some of these are being brought into operation with this number. In the important matter of abstracting ophthalmological literature an agreement has been concluded with the British Medical Association to share in the work they are doing in abstracting world medical literature. All the important ophthalmological publications of the world will be abstracted or noted by the special department of that Association and assisted by our own abstracting service will be made available for us. This service is intended to be fully comprehensive, much more so than was possible for the British Journal of Ophthalmology, and to be up-to-date. As the material available will be greater than can be accommodated in each current number, the British Journal of Ophthalmology will issue, in addition to the normal month's number, a quarterly number of abstracts and it is hoped to include in each of these a summary of new work on some one or other ophthalmological subject. Therefore, henceforth, abstracts will not be published in the regular monthly issues.

Owing to the disruption of life in Continental Europe and the cessation of many specialist publications, authors have been invited to send their papers to the British Journal of Ophthalmology for publication. Many have accepted these invitations and readers of this Journal will find an increasing number of interesting contributions from various foreign countries.

In the matter of illustrations and more particularly colour blocks, it is hoped that we shall be able to print all that are necessary to show clearly the points the authors wish to make. In order to accommodate these it is anticipated that the Journal will have to be increased in size (have many more pages of text) and although paper is still in short supply and its use strictly controlled, it is hoped that sufficient supplies for this purpose will be permitted.

It is obvious that all this will entail a much greater expenditure in the future than in the past, added to this is the increased cost of paper and printing, but the Management feel that our subscribers will prefer to have the much improved journal which is in contemplation. The exact financial implication of all this has yet to be determined and an announcement on this subject will be made later.

"OPHTHALMIC LITERATURE"

An Abstracting Service

In the previous announcement an indication is given that an abstracting supplement will be published quarterly in association with this Journal. To a large extent it will run in conjunction with the abstracting service of the British Medical Association all of whose abstracts are at our disposal, but there will be a considerable number of papers of ophthalmological interest which will be abstracted in our own Ophthalmic Literature, but are not of sufficient general interest to be included in the medical and surgical volumes of abstracts published monthly by the Association. It is very much hoped that our readers will collaborate in preparing these abstracts both for the British Medical Association and for the British Journal of Ophthalmology. We would be glad if those who are willing to participate in the abstracting of ophthalmological papers for the Association would write to the Editor, the Abstracting Service, B.M.A. House, Tavistock Square, London, W.C.1., and those who are willing to abstract such papers as the British Medical Association do not wish to include in their publications would write to Sir Stewart Duke-Elder (Editor, Ophthalmic Literature), 63, Harley Street, London, W.1., stating in either case in which languages they are ophthalmologically proficient in addition to

English. In either case the name of the abstractor will be acknowledged in the text and payment will be made at the rate of thirty shillings per thousand words. Since the literature from January 1, 1947, is being dealt with, an early notification by potential abstractors would be much appreciated.

MR. SURPHLETE, AN ITEM OF OPHTHALMOLOGICAL HISTORY

BY

R. R. JAMES

WOODBRIIDGE

IN Sloane MS. 3801 at the British Museum is mentioned the man whose name heads this paper. In my "Studies in Ophthalmological History" I gave a transcript of the manuscript in question, and may here repeat the lines dealing with Mr. Surphlete.

"Then ther was one Mr. Surphlete a man of axeolente Dyet and crusty fasion of bodye. He lived till he was fouere score yeares of age lived moste in Norfolke & dyed at Linn and in good estate. He lay 2 or 3 yeares at a barber's house at Linn to whom he taught som skille, who nowe professethe it with weak Understandinge and gyven to drinke. I cannot com'end this Mr. Surphlete for any extraordinarye skille though of longe experience."

Mr. Arnold Sorsby has called my attention to the fact that Richard Banister refers to the following oculists in the preface to his Breviary: "Henry Blackborne, famous for the forenamed cures; my kinde acquaintance Robert Hall, of Worcester; Master Velder, of Fennie-Stanton; Master Surflet, of Lynne and Master Barnabie, of Peterborough, all excelling in these operations." He goes on to say that "in their case I noticed much practice but little theorie."

The name Surflet is very rare. Research has established the following facts. A small market town named Surflet is near Spalding in Lincolnshire. A Richard Surflytte married at Maplebeck, Notts., Isabel Lynne on 13, October 1563. A man of the same name (but spelt Surflet), matriculated sizar from Trinity College, Cambridge, at Michaelmas, 1576 (Venn). Richard Surflet, Practitioner in Physicke, translated Andreas Laurentius's "A Discourse of the Preservation of the Sight, and a Treatise of Melancholike Diseases, of Rheumes, and of Old Age." This book was printed by Felix Kingston, for Ralph Iacson, dwelling in Paules Churchyard at the Signe of the Swan, 1599. It is dedicated to Lord La Ware and Ladie Anne, his wife.

Mr. Arnold Sorsby, to whose research I am indebted for my acquaintance with this book, informs me that at the British Museum there are three editions of a book on farming by Richard Surphlet, the last of which is edited by Gervase Markham and was published in 1616.

The book is dedicated to Lord Willoughby, and consists of servile sentiments, classical tags and some Hebrew phrases. Mr. Surphlet's preface, addressed to the "gentle reader," is largely a tirade against the female sex.

Mr. Sorsby tells me that the book itself is composed of extracts from classical authors and is a sort of compendium for farmers. Such human and animal medicine as occurs is largely drawn from the herbals.

Markham's only reference to Surphlet is in the dedication to Lord Willoughby's heir. The previous edition "belonged to your . . . father, as the gift of a learned and well experienced gentleman, who in the translation took a long and well merited labour."

The will of a Richard Surphlet which was proved June 26, 1606, will be given later.

Mr. Sorsby* has suggested to me that the anonymous author of Sloane MS. 3801 may have been Richard Banister himself. I am convinced that he is correct and that I was in error in dating it 1630-40; for in the "Craft of Surgery" by Flint South, edited by Sir D'Arcy Power, I find that on July 8, 1602, Richard Banister of Slyford, Surgeon, was examined and approved by the Court of the Barber Surgeons' Company. It will be recalled that the author of the MS. in question wrote from Sleaford. I think that the man who translated the book of Laurentius may be the Richard Surphlet whose name occurs in Venn. It is possible that he may have been the son of the gentleman who was married in 1563. If the will (v. infra) be his he would appear to have been unmarried and to have left his money in trust to apprentice the most godly of the sons of three other members of the Surphlet family to some honest and profitable trade. These may have been his brothers, though he does not say so. The will which follows is according to the common form of the time; it is chiefly remarkable for religiousness amounting to fanaticism. Richard Surphlet was obviously a Calvinist of the most fanatical type, at the same time he was evidently a scholar, familiar with Greek, Latin and Hebrew. The inventory of his goods shews him to have been rather a dandy, and his library must have been rich in works on divinity and medicine. He was going to sea, possibly to Spain as he took Spanish money with him. At the same time he must have been anticipating cold weather, or he would hardly have noted the stockings "very thick and hayrie, bought at Craconia (Cracow) by a Transilvanian embassadour."

* Vide *Brit. Jl. of Ophthal.*, March, 1934.

I hardly think that Surflet was going to sea as a naval surgeon. At this date James the First had just ascended the English throne; Spain had not recovered from the Armada; and I should surmise that Surflet was simply going abroad. One can imagine the difficulty the executor would have had in deciding which of the sons was the most godly; and if either of them were the ophthalmic quack I think he would have probably been ruled out at once.

I have given footnotes to elucidate the various odd terms which will be found in the inventory, as well as such books as I have been able to identify.

I am obliged to Mr. Harvey Bloom for the transcript of the will.

The Will of Richard Surflet (P.C.C. Stafford, 434)

As I came from the earth the common mother of all mankinde: soe I knowe I shall returne unto the same againe, and with others the workes of God be resolved into my first matter, and seeing the time as a thinge moste uncertaine, is unknown me, but nowe upon this goinge to Sea more probablye approachinge, by reason of the longe and dangerous occurences dependinge thereon. I thoughte it my parte to set my thinges in order, that soo if God doo call me hence, I maye be the freer to wayne myselfe from worldly and transitorie thinges. And to have my mynde onely intente upon these wherein true felicity is placed. And first I bequeath myselfe sowle and bodye to that most loyng and mercifull God who hath given his sonne Jesus Christ, borne of a woman, to suffer death upon the crosse for me and all the elect, nott doubting but that as he hath numbered my dayes and is of all sufficient power to restore me agayne in health and safetie to my Native Soyle (ife soe it seeme good to him in his infinite wisdome) soe he will after my dissolution (whether by land or sea) receive me for the same his Christes sake, into eternall blisse; forgiving me all my synnes originall and actual, imputinge unto me the full and perfect obedience of his deare and onely begotten sonne whereby I shall stande unspotted and blameless before his throane of justice, never to be called into judgement but to passe from death to life; as for the goodes which God of his mercie hath blessed me with all my desire is that they be all sould and turned into money, of the money I give to William Bingham, a carver dwelling in Gleane Alley in Southwark, thre pounce six shillings eight pence. To Maister Travers and Maister Egerton twentie shillings a peece. To Maister Bandeforde and Mr. Paget of Detford tenne shillings a peece. To Mr. Crosley, whom I make Supervisor for the discharginge of this my wyll, and to receive whatsoever moneyes may any manner of way be due unto me, the somme of fiftie three shillings and fower pence. To his wiefe tenne shillings. And to every one of his daughters six shillings eight pence. The Rest of all such moneyes as shall remaine, I desire to be layed out in some lease as may be most profitable either in the Cittie or Countrey. And the yearelye revenue growinge thereofe for the space of every six yeres to be bestowed in the placinge and stockinge of some one of the sonnes of Richard Surflete John Surflete or Robert Surflete suche as shall be ofe most hope for Godlines or towardlines, with some Religious and honest man, in some good and commendable trade; But the Revenue of the seaventh yeare I desire to be bestowed upon the godly distressed and afflicted and this by mutuall intercourse to be contynued from seaven yeres to seaven yeres to the full expiration of such of twentie one yeres, and ife it should please God to call Maister Crosley away by death before such tyme expired. Then my desire is that he would commende these partes and offices to be performed by some other honeste frende of his fearinge God, or else to gett Maister Travers or Maister Egerton or some other faithfull minister such as shall then be of good reporte like unto them here in the Cittie to procure one to doe the same to which my last wyll and Testament made the first of March one thouzand six hundred and thre I putt my hande and seale in the presence of us Hugh Evans, William Tilney, William Shambrooke, Abraham Webbe By me Richard Surflete:—Leit in the Cytue.

An Inventorye of such goodes as I carie with me to the Sea or otherwise leave

with my friendes in London made the day and yeare within wrytten. Imprimis one great vessell of bookes at Maister Theralde the Lynnen Draper his house in Fryday Streete at the Signe of the Maydens head and Unicorn, and in the same place one old hampear with nothing but trash. Item at Maister Crosley his house at the Goulden Lyon at the Stockes one hamper and in it a clock called an alarum, a great box with Surgery instrumentes a lesser box called a playste or box with surgery instrumentes, also some of silver and some of iron, a box with my letters of orders, a license to practise Physique my clocke plummettes and a canvas bag with sundry sortes of thinges in it. Item one rapyer and a dagger a grograine gowne¹ with a velvet face cape and gürding, a velvet jerkin, a satten doublet. two stuff doublets, two paire of rounde cloth hose, one paire of hose of velvet panes and cannyons,¹¹ one paire of new Jersie stockings of fourtene shillings pryce, one peece of dornix² with curtaine ringes at it, one ball of velvet gürding, one newe clarke* faced with velvet, one rydinge clarke and an old paire of baces,³ one paire of bootes and spurres one cloth saddle with three girthes a petterell⁴ and crupper, one bridle with bytt and bosses, six ruffe bandes of cambrick, one new hatt faced with velvet, one course sheete to wrap my apparrell in, one velvet muff, one velvet cape for a clarke upon cloth.

Taken with me to sea First one Truncke, one mayl thre boxes with lockes whereof one is made deskwyse, two gównes the one of stuffe the other of cloth, one clark faced with velvet, one hat one Dutch cap, one silcke quilted cap, one other cap of Sparta velvet, two fustian nightcappes seaven lynnen cappes, whereof one is a lyttle wrought with black silcke in a border about the edges and thre of the other are newe, five dozen and a halfe of falling bandes two dozen new hande towelles and as many tablenapkins handkerchers, two dozen and a halfe cufes thre newe paire with black and white edging lace, and as many payres of old ones, two doubletes of stuffe, fower jenkins, one of silcke another of cloth, the two other of stuffe, one newe the other olde, two crane feathered fustian mandillions,⁵ two paire of drawing breeches of the same, fower paire of clothe breeches, two trusses of canvas six paire of lynnen breeches six fustian wastcoates, eightene newe shirtes, and six old, thre paire of oiled leather lynynges five paire of lynnen stockings whereof fower are newe, one paire of worsted stockings blacke and newe, two paire of Jersey but old, a fowerth paire of woollen yarne newe, a fife pair of russet cloth, and a sixte of whyte knytt ones very thick and hayrie, bought at Craconia⁶ by a Transilvanian Embassadour, newe shoes three paire, old shoes two paire one paire of pumppes and pantofles⁷ olde, a paire of slippers and olde paire of lyned moyles, but lyttle or nothing worne, one paire of newe Pennynston⁸ blankettes, three newe paire of hose garters, two of crewell and one of silcke, one bedrugge newe, one woolbed and two pillowes, two fustian pillowberes⁹ newe, one mat, one brush for clothes, one headbrushe, a lookinge glasse, two combes of box in cases, a paire of snuffers, an extinguisher a wooden standish¹⁰ with penknife etc a fayre Inckhorne with a penner and sandbox two, and of the same, fower newe paire of gloves, an old motley clokebagge, bookes a hundred and eight or thereabouts and of name as followeth, one hebrew bible one Tremelius in fol. one English bible guylded and prynted at Geneva, a French bible, one Greeke Testament, one French Testament, Calvine on Deuteronomy and Job, Calvine upon the Psalmes in Latin, one Institution and epistles, Beya his epistles, and confessions, Du Plessy against the Masse, Grinous workes Parkins¹² workes fol. Daneus on the Lords Prayer his ethicks and Isagoge *Christiana* in 2 vol. One Polain *Theses*, and *Theses Genouses* one Perkin's¹¹ problem, his booke, *de predestinatione his prophetica* and *Specimen degestri*, one Ursinus *Catechisme Sculteti medulla patrum, Flores Doctorum*, one small Tablebooke the drum of doomesday. Kewstubbes¹¹ on the Commandementes. Deanuge's lectures, Virel's dialogues, Bakers¹³ lectures on the Creede, the harmony of confessions. Fenner's¹⁶ *Theologia*, the Councell of Trent. One Perkins of Sathan's sophistry. Hunter's *Cosmographie*, one sermon of repentance six small catechismes the Goulden booke of the leaden Godes. Brocard on the Cantacles. Finch his divynitie, one treatise of fastinge set forth in Scotlande, one Immytacon of Christ. One forme of comon prayer, one monomachie of motives, Cartwright's¹⁷ little Catechisme wrytten, one other booke of the forme of common prayer etc, one other Catechisme with the text of the Quotations, Stephanus his concordance Fenners household discipline one destruction of smale vices, one other smale Catechism. Fornelius¹⁴ his workes fol. Piso his methodus etc. Valverda¹⁹

* Cloak

his Anatomical tables, Bannasters²⁰ Anathomy, Weckers²¹ special Antidotane, Parry²² his surgery, Schenkens²³ his observations 2 vol. Penotus²⁴ *de vera preparatione medicamentorum chymicorum*, The Cure of diseases in remote Regions, Gratias *ab horto* of East Indian Simples. Laurentius²⁵ de Cresibus, Clowes²⁶ his practise of Surgery, Botellus²⁷ *de missione sanguinis*, Willichus²⁸ *de Urinarum probationibus* Wecker's surgery in English, Rouseus²⁹ *de magnis hippo-tenib*, one great lexicon, one Thom: dictionary one *dictionary poeticum*, Martinus Gramer, Paginus Epitome, Scotus Greeke gramer, Mathiolus of the facultyes of simpies. decimo sexto, one Taleus Rethorick, Cartwrightes Treatise of Fastinge, Phregius his pedagogus one Licesthenes Apothegmes, Aristotles Sentences, Tullyes Sentences, *Flores poetarum*, the small Greeke poets. Verons Phisicke, Daneus his Physicke, Ovids Metamorphosis, three small paper bookes. Bourne his Regiment for the Sea, one bounde paper booke conteyning one quire folio, paper booke conteyning one Epitome of Mr. Cartwright uppon the Commandementes and twentie poundes in Spanish Ryalles

Commission to administer was granted to William Crosley, June 26. 1606.

A search of the Parish Register of King's Lynn by the Verger gave the burial of the daughter of a physician there in 1630, which seemed at first sight as if it might be a name like Surphlet; but the tracing which he sent makes it more likely to have been Curtis:—"Margaret Curtes d. of Mr. Richard Curtys, a physitian."

I do not of course pretend that this paper settles the question as to who Mr. Surphlet, the quack oculist was. The testator may have been the translator of Laurentius, but it is odd that he did not mention his own work in his inventory. We know that the quack died at Lynn; and it is to be assumed that the testator lived in London or Southwark. He probably died abroad, and proof of death must have been forthcoming for administration to be granted to his executor.

The will is of interest in many ways and partly as shewing what a medical man at that date thought fit to take to sea with him.

FOOTNOTES

1. A red silk gown.
2. Usually spelt Dornick, a species of linen table-cloth, used in Scotland, and originally made at Deornick, in Flanders.
3. Probably braces.
4. A horse's breastplate.
5. A soldier's coat.
6. Cracow, capital of Poland.
7. Slippers.
8. Possibly Penistone in Lancashire.
9. Pillow beer, a cover for pillow-case.
10. A writing desk for ink, sand, wafers, etc.
11. Panels and lappets.
12. William Parkins (1558-1602). A staunch Calvinist whose works were translated into Dutch, Spanish, Welsh and Irish. The most famous, his *Armilla Aurca* (1590) went to its 15th edition in 20 years. *Dict. Nat. Biog.*
13. *Vide supra* Parkins.
14. Possibly John Stubbs or Stubbe, a puritan Zealot. (1543-1591). *Dict. Nat. Biog.*

15. Possibly Sir Richard Baker (1568-1645), Religious and Historical writer. *Dict. Nat. Biog.*
16. Dudley Fenner (c. 1558-1587) Puritan Divine. His *Sacra Theologia* was published in 1585. *Dict. Nat. Biog.*
17. Thomas Cartwright (1535-1603). A Puritan. *Dict. Nat. Biog.*
18. Jean Fernel (1506-1588). His *Universa Medicina* was published in folio in 1567.
19. Valverde de Hamusco. His Anatomical Tables (Rome, 1556) are well known.
20. John Banester (1533-1610).
21. J. J. Wecker, the author of *De Scerctis*, 1582. *Antidotarium, generale et speciale*. 1601.
22. Ambroise Paré.
23. Schenckius, James. Observations. *Lib. VII. de Veneris*. 1597.
24. Penotus. 1602.
25. Laurentius, on Crises.
26. Clowes, the well known English surgeon. Surgeon to St. Bartholomew's Hospital.
27. Leonardus Botallus. *Dr. Curatione per Sanguinis Missionem*. 1583.
28. Willichius. *Urinarum Probationes*. 1582.
29. Rouseus was the author of some works but I have not been able to identify the one mentioned here.

REMOVAL OF THE WRONG EYE *

BY

H. M. TRAQUAIR

EDINBURGH

THE most terrible disaster which can occur to the ophthalmic surgeon and to his patient is "removal of the wrong eye."

It might be thought that this accident was merely a possible but unlikely danger about which teachers should warn students but which has never actually occurred. That was my impression when I wrote in 1916¹ that by the use of local analgesia "the possibility, however remote, of such a calamity as its (the wrong eye's) removal is totally avoided." At that time I imagined that removal of the wrong eye was a hypothetical possibility rather than an actual fact, a view which had been to some extent previously expressed by Hermann Knapp² when he wrote in 1898 " . . . we should be on our guard lest we take the good eye out. This awful mistake is sensationally mentioned in text-books and periodicals; I do not know whether it has actually occurred, but the possibility is undeniable."

Knapp's statement was based on the literature before 1898. Of over sixty text-books on ophthalmology and ophthalmic surgery

* Received for publication, December 4, 1946.

published before that year I have been able to examine thirty-seven in only one of which (Mauthner¹) is the subject mentioned. Only four of fourteen books published since 1898 contain references and also two later editions of older works (Lawson³, Czermak⁵). Recent works do not mention the subject, the Graefe-Saemisch Handbuch⁹ (Sattler, 1922), appears to be the latest. Medico-legal text-books are likewise silent. There cannot have been many "text-books and periodicals" in which Hermann Knapp found the subject "sensationally mentioned."

It may be noted that of eight direct references only one is British, the remainder being German and American.

There is, nevertheless, abundant evidence that this catastrophe has actually occurred. Apocryphal stories exist in relation to both eastern and western hemispheres; a well-known one is that of the surgeon, who on discovering his mistake rushed into an adjoining room and shot himself. According to another the patient received a *solatium* of £10,000 and a pension. It is related that in a case (presumably of glioma) in a child, the surgeon, having removed the wrong eye, promptly removed the other one also and explained to the parents that bilateral removal was necessary as the disease always affected both eyes. In this instance, however, it is within the bounds of possibility that the surgeon's error saved the patient's life. Such stories have no value as evidence, their only interest lies in the suggestion that where there is smoke there is probably fire.

The scanty references in the literature mostly contain warnings against the risk of the accident and advice as to how it may be avoided. The method recommended is the indication of the eye to be removed by affixing a piece of adhesive plaster on the brow or in some other way such as by bandaging it. The earliest reference I have found is that of Mauthner¹ who wrote in 1881 that he had been personally present when the mistake was *nearly* made in a case of sympathetic ophthalmia. His own words are worth quoting:—

"Bei der Enucleation, wie sie auf dem Gebiete der sympathischen Leiden kommt, ist aber Eines die Hauptsache, und dies ist, dass man das richtige Auge enucleirt. Das scheint ein müssiger Rath, vielleicht ein Scherz, aber wer wie ich schauernd dabeigestanden, wie statt des erblindeten Auges bald das noch sehende enucleirt worden wäre, scherzt nicht bei diesen Worten. Das Versehen ist nicht so unerklärlich, wenn man bedenkt, dass die Enucleation ja so häufig bei schon entwickelter sympathischer Kyklitis ausgeführt wird, dass im Aussehen der beiden Augen nicht immer ein markanter Unterschied bemerkbar ist, und dass der Operateur, sein ganzes Augenmerk auf die Operation richtend, sich willig vom Assistent leiten lassend, die Operation an jenem Auge beginnt, in welches der Assistent irrthümlich die Lidhalter

eingelegt. Der Patient rührt sich nicht, denn er ist - - - narcotisiert.”*

Elschnig⁵, re-editing Czermak's book on ophthalmic operations, quotes Mauthner and adds the following passage:—

“Dasselbe konnte aber, und vielleicht noch leichter, vorkommen wenn es sich um einen intraokulären Tumor handelt, der noch zu keinen Veränderungen am vorderen Augapfelschnitt geführt hat. Da sehen beide Augäpfel ganz gleich und normal aus. Man gebe sich also vor Beginn jeder Enukleation Rechenschaft, welches das zu enukleierende Auge ist, und in Fällen, wo sein Aussehen nicht sicheres Merkzeichen trägt, verlasse man sich nie auf sein Gedächtnis, sondern sehe unmittelbar vorher in seiner Vormerkung nach. Das mag manchem als überflüssige Vorsicht erscheinen, allein beim besten Gedächtnisse *kann einmal ein Irrthum unterlaufen*, und wenn er in einem derartigen Falle unterliefe - - - ist es überflüssig das auszumalen.

(Ein Irrthum ist jedenfalls dann ausgeschlossen, wenn das zu enukleierende Auge vor Beginn der eventuellen Narkose gereinigt und mit einem Heftpflasterbande geschlossen wird.)

(Soll übrigens seither schon tatsächlich vorgekommen sein!).”†

The interesting point in Elschnig's statement is contained in the footnote which shows that, evidently in spite of precautions, a case had occurred within his knowledge.

Haab⁴ mentions the danger associated with a general anaesthetic and points out that both eyes may be abnormal in appearance though equally so, as in sympathetic ophthalmia. This condition, however, as a factor in connection with removal of the wrong eye is of little

* “In enucleation, as practised in sympathetic affections, it is of chief importance that one enucleates the correct eye. That may appear superfluous advice, even a joke, but one who like myself has stood horrified while the seeing eye was nearly enucleated instead of the blind one will not find amusement in these words. The accident is not so unexplainable when one remembers that enucleation is so often performed in already developed sympathetic cyclitis, that there is not always a pronounced difference in the external appearance of the two eyes, and that the surgeon, all his attention directed towards his operation and willingly allowing himself to be guided by his assistant, begins the operation on the eye in which the assistant has mistakenly inserted the speculum. The patient does not disturb himself, for he is . . . anaesthetised.”

† “The error can even more easily occur when dealing with an intra-ocular tumour which has not caused any changes in the anterior segment of the eye. Both eyeballs appear normal and the same. Before beginning any enucleation one should therefore provide oneself with a statement as to which eye is to be removed, and in cases where no definite external sign is present one should never rely on one's memory but should refer to notes immediately beforehand. That may seem to many a superfluous precaution but even with the best of memories it is possible for an error to slip in, and if in such a case it should occur - - - it is unnecessary to say more.

(A mistake can, in any case, be excluded if, before commencing the anaesthesia, the eye to be removed is cleaned and covered with an adhesive plaster).

(Since then a case has already, after all, actually occurred!).”

importance nowadays as the offending eye is usually removed at the earliest sign of change in the other or before any sign has appeared.

Beard⁶ writes "That this most deplorable accident is not beyond the realm of the possible has been abundantly proven and that by more than a single instance."

Wood⁷ says "Lamentable but, fortunately rare instances have occurred....."

"Near misses" (Mauthner¹, Lawson¹⁰) are relatively more common. In these the operation is commenced or about to be commenced on the wrong eye, or wrong side, but the mistake is discovered before serious harm has been done. Conversations with colleagues indicate that many have had experiences of this nature.

There is much evidence that paired or multiple organs can easily be confused. Burrows¹¹, writing of operating on the wrong side for hernia states "Such confusion between the left hand and the right appears to be no rare thing in clinical note, taking;....."

Here the error is in the notes rather than in failure to refer to them. The wrong eye has been operated on for cataract, the wrong kidney and the wrong finger have been operated on, and the wrong leg has been amputated (for sarcoma). Innumerable wrong teeth have been extracted: the present writer has made this mistake himself.

For the removal of the wrong eye two postulates are essential:—The patient must be under a general anaesthetic and the eye to be removed must not be obviously different in appearance from the other. Secondary or adjuvant causes include omission to indicate the eye by a mark on the forehead or even delegation of the marking to a nurse or house surgeon, an error in the notes, or omission to verify the eye to be removed by examination or reference to notes immediately before the operation. The surgeon may hurry into the operating theatre where the patient is waiting on the table completely anaesthetised and proceed without delay or further scrutiny to remove "an" eye.

It is evident that advice to mark the eye and the other precautions mentioned, which have been recommended during the last fifty years and are included in the teaching of many ophthalmic surgeons, have not produced the desired results as cases have occurred within recent years. It will, presumably, never be known except to those directly concerned whether the mistake has ever occurred owing to marking of the wrong eye or an error in the case notes but such possibilities constitute the weak point of reliance on any safeguard other than immediate pre-operative examination of the patient in cases in which a general anaesthetic is used.

The diseases concerned, in actual instances, have been intra-ocular tumour, glaucoma, and sympathetic ophthalmia, probably mainly the first. Nothing is known as to even the approximate number of cases. As might be expected, in every known case a general anaesthetic was used.

In infants and young children the risk must be taken and it is the duty of the surgeon to take proper precautions personally, and not to delegate such an important responsibility. The eye should be examined immediately before the operation while the patient is on the table. If a general anaesthetic is to be given either to an adult or to a child administration should not be commenced until the surgeon is present.

The only infallible preventive, if any measure can be infallible, is the use of local analgesia. This method should be adopted in all adult cases in which the eye to be removed is not obviously and distinctly different in external appearance from the other.

A paragraph on this subject should be included in every text-book in which removal of the eye is mentioned.

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THE INTRA-OCULAR FOREIGN BODY

A Series of 72 Cases in the B.L.A.

BY

H. B. STALLARD

LONDON

THIS paper is an account of 72 cases of penetrating wounds of the eye with retained intra-ocular foreign body which came to a field hospital in Normandy and a 2,000-bedded General Hospital in Belgium (B.L.A.) from July, 1944, to the end of hostilities in Europe in May, 1945. It is the sequel to a report of 102 such cases, treated from the beginning of General Cunningham's offensive in the Western Desert (M.E.F.) in November, 1941, to the end of

the North Africa campaign in May, 1943, which was published in the *Brit. Jl. Ophthalm.*, Vol. XXVIII, p. 105, 1944.

As the following statistics show, these cases include besides battle casualties accidental injuries in field workshops, and injuries inflicted by foolish and negligent handling of military weapons.

The majority of foreign bodies produced by the fragmentation of modern war missiles are so lowly magnetic that the importance of more accurate localisation than is necessary in civil practice soon became evident to us for it was essential to bring the terminal of a giant electro-magnet as near to the intra-ocular foreign body as possible in order to effect its extraction. When this was done in a number of cases in which the foreign body had been labelled as non-magnetic in other medical units it was extracted. Probably the fault in these cases lay in relying on the so-called "magnet test" in which pain is elicited on applying the magnet to the eye. Either the foreign body when attracted to the magnet moved a certain distance but did not quite reach the retina and choroid (a fact sometimes seen when the foreign body could be followed with an ophthalmoscope whilst the terminal of a giant-magnet was brought as near as possible to the sclera) or the impact was too gentle to elicit pain, or as it seemed in some cases the sensation of

TABLE A

Route of extraction	M.E.F. 102 cases			B.L.A. 72 cases		
	B.C.	Acc.	Total	B.C.	Acc.	Total
Posterior (Scleral) route	22 (21.5%)	9 (8.8%)	31 (30.3%)	32 (44.4%)	5 (6.9%)	37 (51.3%)
Anterior route	5 (4.9%)	3 (2.9%)	8 (7.8%)	9 (12.5%)	4 (5.5%)	13 (18%)
Total ...	27 (26.4%)	12 (11.7%)	39 (38.1%)	41 (56.9%)	9 (12.4%)	50 (69.3%)

Table A shows the percentage of intra-ocular foreign bodies extracted by a giant electro-magnet through (1) the scleral route (2) the anterior route.

M.E.F.—Middle East Force.

B.L.A.—British Liberation Army.

B.C.—Battle Casualty.

Acc.—Accidental injury.

the intra-ocular membranes was temporarily impaired by either contusion or concussion changes. An even more probable cause of failure was the use of a giant electro-magnet terminal at a distance too remote to move the intra-ocular foreign body to a position where it could be either felt or seen. Particularly was this so when attempts were made to bring forward a foreign body from the vitreous in the first stage of the anterior route technique for extraction.

Table A shows that with improved equipment and proper technique the successful extractions of intra-ocular foreign bodies were increased. Although the type of war missiles was much the same in both theatres of war the figures for Normandy and Belgium (despite working under crude field conditions in Normandy) are better than those of the Middle East Force.

It is possible that in future with an improved magnet the number of successful foreign body extractions by the scleral route might be increased.

Investigation

A reliable diagnosis can only be made with a good light such as either the Lister or scialytic operating lamp, surface anaesthesia with pantocaine 1 per cent., a Desmarres' retractor, binocular loupe, the slit-lamp and corneal microscope, ophthalmoscope, Schiötz tonometer (in some cases) and radiography. It is essential to examine carefully the adjacent anatomical structures bounding the orbit, the nose, face, and skull; particularly is this so in military surgery, where a missile in its course may have damaged the face, accessory nasal sinuses, eye, orbit, and intra-cranial contents. In one case a fine shell splinter had penetrated the occiput, traversed the intra-cranial contents, entered the orbit from behind and penetrated the sclera to come to rest in the vitreous. The small scalp wound had healed and the soldier was unaware of its existence. In another instance the intra-ocular foreign body had traversed the right eye (a through and through scleral wound) the right os planum, nasal septum, left os planum and through the sclera on the nasal side of the left eye. The general condition of the patient must be considered, haemorrhage from other wounds and shock requiring first attention. It is surprising how little the majority of eye wounds suffer from being left alone after simple cleansing of the eye and lids and covering with a pad and bandage. In the Western Desert and North African campaigns in 1941, 1942 and 1943, delay in evacuation of casualties through the field ambulances and casualty clearing stations amounted in some instances to 5 to 12 days before the soldier with an injured eye reached an eye

surgeon. It was remarkable to see many such eyes with little or no clinical evidence of inflammation.

However, in the case of intra-ocular foreign bodies prompt surgical attention is desirable in principle. Delay may allow the foreign body to become entangled in a collection of fibroblasts, and a mass of surrounding exudate, possibly infective, may jeopardise other intra-ocular tissues by trailing the foreign body in the course of its extraction.

An entry wound in the sclera may be difficult to find when it is only a millimetre or so long and has healed by the time the patient is seen. In military surgery a guide to its position may be given by a snick in the lid margin, a wound in the eyelids and the skin adjacent to the orbit. With the slit-lamp and corneal microscope the bulbar conjunctiva is seen adherent and puckered at the site of the scleral wound. In grosser cases a small knuckle of herniated vitreous covered with young fibrous tissue and conjunctiva is sometimes evident.

The scleral wound may be behind the equator and the reflection of the bulbar conjunctiva and so will not be seen until Tenon's capsule is opened in surgical exploration of the injured site. In such cases the lens is often clear, unless the foreign body has passed obliquely forwards and struck it, and so ophthalmoscopic examination will reveal the site of the entry wound unless this is obscured by a severe intra-ocular haemorrhage.

Aluminium and some non-magnetic alloys give rise to no apparent signs of irritation inside the eye and when small are best left alone. Larger non-magnetic foreign bodies causing visual obstruction and likely to cause intra-ocular inflammation may be removed by an instrument introduced into the eye when the foreign body can be seen by means of the ophthalmoscope and the instrument thus guided to it under view. Small fragments of glass and bakelite may remain quiescent in the eye for years but some, particularly when situated in the filtration angle or on the iris, cause irritation and require removal by forceps, blunt hook or scoop. Copper and stone cause rapid intra-ocular inflammation in most cases and evisceration becomes necessary. There are, however, some cases of soldiers who retained multiple minute particles of stone on the iris and in the cornea and sclera following a land mine or booby trap explosion whose eyes settled down after several weeks' or months' irritation and have remained quiet. Possibly the fragments were rendered sterile in the explosion and so are unlike those seen in civil injuries, which often become infected.

Important facts are the likely nature of the missile striking the eye, the force and direction from which it came, and the position of the patient's head when the foreign body struck him. In war

wounds fragments of metal removed from other parts of the body are tested by a magnet and so afford useful information as to the probable character of the intra-ocular foreign body.

Localization of the intra-ocular foreign body

1. *Ophthalmoscopic*.—In 17 out of 102 cases of intra-ocular foreign bodies from war missiles in the Desert Campaign 1941-43, in 28 out of 72 cases in the fighting in France, Belgium and Germany 1944-45, the foreign body was seen by the ophthalmoscope. In 21 of the latter it was extracted by the posterior route (through the sclera), 19 by the giant electro-magnet and 2 by forceps under ophthalmoscopic control. Three were extracted by the anterior route. In the remainder the foreign bodies were non-magnetic. These were less than 0.5 mm. in most instances, showed no evidence of adjacent inflammation, and so were left.

The ophthalmoscope is also of great use, in cases where the media are clear and the foreign body can be seen in the vitreous, for finding out whether it is magnetic or not and the excursion it will make on application of the magnet terminal to the sclera at the nearest accessible point to it. If the foreign body reaches the retina on applying the magnet then it may be extracted through a scleral incision. In some cases, particularly those of very small foreign bodies, 0.25 mm. or less, the foreign body may move only a few millimetres in the vitreous and not reach the retina. In some cases postural treatment and repeated applications of the magnet may eventually make it accessible for removal. In 8 instances the foreign body moved between 1 and 4 mm. and did not reach the retina, and in 6 of these it was extracted by the magnet after opening the sclera, choroid and retina.

It is important not to be misled by the floating movement of a small particle of a light metal suspended in the vitreous which alters its position with movements of the eye on changing the position of the head. Before applying the magnet the position of the foreign body should be carefully noted for this may be different in the lying and sitting positions. The magnetic movement is quite unmistakable, it is a sudden jerk towards the magnet.

2. *Radiographic*.—Radiographic localisation of an intra-ocular foreign body is essential in every case of injury due to war missile even though the foreign body is evident on ophthalmoscopic examination, for X-rays may reveal the presence of other foreign bodies. It is, however, not an infallible test for in a number of cases X-rays failed to reveal small foreign bodies of less than 1 mm. which were seen with the ophthalmoscope, were magnetic and extracted through the scleral route.

There are about 30 methods of X-ray localisation of intra-ocular

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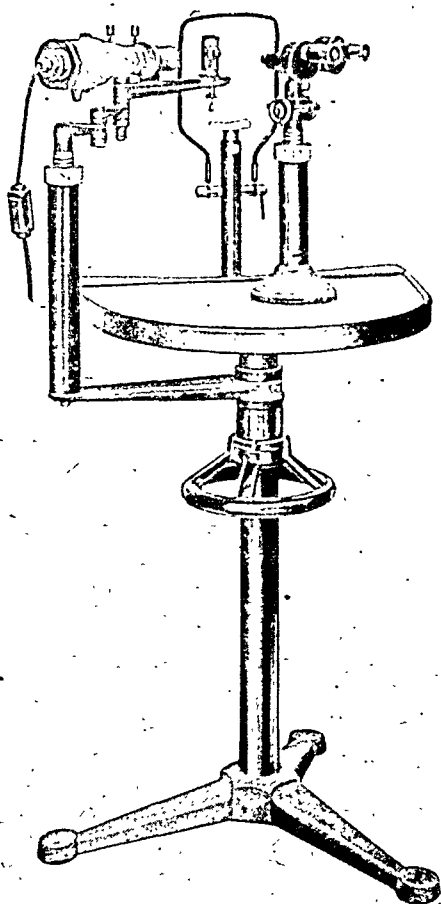


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foreign bodies. These have been classified into 6 groups in Vol. III of "A Textbook of X-ray Diagnosis" by British Authors and also are fully described in "La Radiographie en Ophtalmologie" by E. Hartmann.

Perfection in the radiographic localisation of an intra-ocular foreign body has not yet been reached. The technique selected often depends upon the nature of the case and the apparatus available.

Under ideal conditions there should be a device for keeping the head absolutely immobile, the provision of an occipital rest and a horse-shoe shaped bar covered with soft dental stent wax for the patient to bite achieves this purpose. Immobility of the eye during exposure to X-rays is effected by the provision of a suitable target, such as a black cross on a white background for the patient to look at. Several such targets are necessary when views are taken of the eye looking in front, up, down, to the nasal and temporal side. The patient is more comfortable either lying on his back or sitting up. Some positions such as lying face downwards and looking into a mirror at close range (Comberg's technique) are awkward and painful. Multiple wounds necessitate the dorsal position and the radiographic technique must be arranged accordingly.

The more elaborate and expensive instruments such as those devised by Sweet and Dixon have a radio-opaque indicator mounted at a known distance (10 mm.) from the centre of the cornea. Additional aids in other procedures where simple apparatus only is available are the attachment of radio-opaque markers to the surface of the eye; these, such as little silver clips or hooks, may be inserted into the conjunctiva or sewn into place such as the limbal ring described below. Skeoch's scleral ring and the corneo-scleral contact glass with either radio-opaque dots or a ring as used by Comberg and Wessely are placed free in the conjunctival sac. The disadvantage of the attachment of either clips or a limbal ring is that a surgical procedure is necessary to effect this. Skeoch's scleral ring and the contact glass are both liable to slip out of position and tilting and moving of these devices is appreciable when the eye is turned in different directions. An error in localisation is likely to occur in such cases. An opaque marker may obscure an intra-ocular foreign body in the antero-posterior view but the latter will be evident in the lateral view. The various methods employed in the localisation of an intra-ocular foreign body may be classified according to the main principle in the technique. These are six.

(1) *Physiological*.—In this method the head is fixed and the eyes are moved in prescribed directions. Some useful information may be obtained by calculations of the movement of a foreign body

on movement of the eye. Two to four exposures may be made on the same film, the patient being directed to look up, down, to the nasal and temporal sides. The eye is sub-divided into 4 quadrants in front of the equator and 4 behind it. The movement of the foreign body will be with the eye in the anterior quadrants, and against it in the posterior quadrants. The centre of rotation of the eye is never a fixed point so that the theoretical assumption that a foreign body at this point would not move does not hold. The surgeon is chiefly interested in the relation of the foreign body to the sclera.

Belot and Fraudet centre the X-ray tube at a point they judge to be the centre of rotation of the eye and take three lateral views looking up, in front and down, and three anterior views looking in front, nasally and temporally.

The difficulties in this method of localisation lie in centring the X-ray tube accurately and in keeping the patient's head absolutely still during the movements of his eyes.

If the foreign body is well clear of the eye in the orbital tissues there is no movement. Displacement sometimes occurs in the orbital fat, a heavy foreign body such as a lead pellet being pressed to another position when the eye is moved.

Movement of an extra-ocular foreign body may also occur when it is situated in Tenon's capsule, an extra-ocular muscle and in the optic nerve. When it is in the bulbar conjunctiva it will of course move but it is visible unless obscured by a subconjunctival haemorrhage. Foreign bodies in the lids also move.

In war injuries the multiplicity of foreign bodies in the orbit make an accurate diagnosis of the number and site of those in the eye very difficult.

(2) *Geometric*.—In this method the head and the eye are immobile, the X-ray tube is moved and views are taken from several known angles with a radio-opaque indicator 10 mm. in front of the centre of the cornea. The calculations are plotted on a chart. Sweet and Dixon use this technique and probably it is the most accurate of all methods. The apparatus is elaborate and expensive, only a few hospitals possess it and it was never available in field medical units in a theatre of war.

Kraus and Briggs claim greater accuracy with their apparatus than any other, the essentials of which are (1) head rest with clamping devices for immobilisation of the head, (2) attached to the forehead cross-bar is a small unit designed to work to fine adjustment and carrying indicators which may be moved laterally, up and down and to and from the eye. These indicators are made partly of radio-opaque and partly of non-radio-opaque materials; (3) X-ray cassette holders and perimeter with spot-light.

The indicator, axis of the eye and centre of the X-ray tube must

be in one straight line. The tube (central ray) must not be tilted or distortion will occur. The best anode to indicator distance is probably one metre, at which the magnification will be negligible (1 : 20). Exposure depends on the X-ray apparatus used. For each exposure one of the indicators is placed in contact with the cornea at the centre of the pupil and the scale attached to it will show any magnification.

The calculation of the site of the foreign body is a complicated geometric problem and Stern comments that in this instance it is fallacious and that the simpler limbal ring method gives as accurate results.

(3) *Stereoscopic*.—The principle is the same as the geometric method. One exposure is made with the head in the prone position and inclined toward the affected eye so that the X-ray tube aimed at an angle tangential to the lateral orbital margin may obtain one exposure with the minimum of bone shadow. Another view is taken laterally with a contact glass and radio-opaque ring embedded at the limbus, then 3 successive photographs on 3 different films taken of the eye looking up, in front and down.

This method does not give precise measurements of the distance of the foreign body from the ocular tunics.

(4) "*Simple*." — Hartmann includes in this group X-ray measures which use a radio-opaque marker in contact with or close to the eye and require only simple X-ray apparatus. The circumstances of military surgery in the field necessitate the employment of such methods, for neither elaborate apparatus nor radiologists experienced in this special work are available.

It is therefore imperative to have some simple procedure which may be done expeditely by the eye surgeon and the X-ray department staff operating under crude conditions in a tent.

A brief account of some of the radio-opaque markers has been given above. Of these the limbal ring, Comberg's and Wessely's contact lenses and Skeoch's scleral rings are worthy of mention. The chief advantage of these devices is that by their close contact with the eye they afford greater accuracy in measuring the distance of the foreign body from the known point where they lie against the eye.

The movement of Comberg's and Wessely's contact glasses containing 4 radio-opaque dots or a ring at the limbus and the tilting of Skeoch's scleral ring are causes of inaccurate calculations. Efforts have been made to secure immobility of the marker by suturing it to the conjunctiva. Thorpe (H. E.) had Comberg's contact lens drilled with 4 holes, 3 of these were placed near the periphery of the scleral part of the glass at 3, 6, and 9 o'clock respectively for the purpose of suturing to the bulbar conjunctiva. He made also a fourth hole at the limbus in the 1.30 o'clock

meridian so that air could reach the cornea. The bulbar conjunctiva more than 3 mm. from the limbus is mobile so that suturing here does not give absolute immobility to a contact glass.

Another authority has fashioned a projecting boss from the centre of the corneal part of the contact glass so that by seizing this with forceps he may adjust the position of the glass with the opaque limbal ring directly over the limbus immediately before X-ray exposure.

Yazujiam's combined limbal and scleral ring, is I think unnecessarily elaborate. It consists of the limbal ring of 12 mm. inside diameter, joined by 4 radiating bars of 6 mm. length with the scleral ring which has 22 mm. inside diameter and 24 mm. outside diameter. The cross bars may obscure the foreign body in both the lateral and antero-posterior views.

Skeoch's ring is made of stainless steel dental wire gauge 30 and welded with a lead bead. There are 3 sizes of internal diameter 24, 25 and 26 mm. respectively. The ring is slid into the upper and lower fornices with the lead bead just above the caruncle. The equatorial fit is checked by directing the patient to look to the right and then to the left.

Three X-rays are taken (1) lateral (2) oblique lateral. The ring tilts and the eye moves twice as far as the ring. (3) postero-anterior. The lead bead and ring afford shadows against which the density of the foreign body is compared.

The limbal ring.—Of all these marking devices the limbal ring, described by A. C. Norman in 1915, gives results which are reasonably accurate and of practical value in the majority of cases. The rings are made of silver wire 1 mm. in diameter and sizes varying in the internal diameter of the ring from 9 to 13 mm. are used. The ring which exactly fits the limbus is chosen and its immobility on the eye is assured by suturing it at 9, 12 and 3 o'clock to the conjunctiva at the limbus. For 3 mm. or so behind the limbus the bulbar conjunctiva is firmly attached to the episcleral tissues and does not ride easily over the sclera as is the case with the bulbar conjunctiva behind this zone.

A refinement which is, I think, helpful in the orientation of the eye is to fuse to the limbal ring 3 small silver loops at 9, 12 and 3 o'clock for the passage of the suture and the marking of these meridians, any deviation of which would be noted in the X-ray film and allowed for in assessing the meridian in which the foreign body lies. The technique is as follows :—

The eye is anæsthetized with pantocaine, and when much inflamed 2 or 3 minims of novutox are injected into the episcleral tissues at the limbus at 12, 9, and 3 o'clock. A silver ring of 1 mm. thickness and of a size chosen to fit exactly the corneo-scleral junction is stitched in position by sutures of 00 silk passing

through the conjunctiva at 12, 9 and 3 o'clock respectively. A drop of ol. parolein is instilled into the eye and a pad moistened in the oil is applied to the closed lids and bandaged in position. When possible the patient sits during X-ray examination. In some cases other wounds prevent this. During the first exposure he is directed to look with his uncovered eye forward, and for the second to look downward. For this purpose conspicuous marks such as red crosses or lights 5 cm. in diameter are placed on the wall for a sitting patient and on the ceiling for lying cases at the two points in which fixation of the eye is necessary during exposure. Each exposure is half the normal. To obtain a postero-anterior view of the orbit free from the dense shadow of the petrous part of the temporal bone the head is tilted slightly so that the occiput is down and the face up; the petrous shadow then falls over the antra. In an accurate postero-anterior radiograph the silver ring shows as a perfect circle; and in the lateral as a linear shadow. To obtain a perfect linear shadow in the first position of the lateral view Captain V. Lees, R.A.M.C., suggested that a mirror be attached to the X-ray apparatus in front of the unaffected eye and exactly at right angles to the X-ray cassette holder. The patient is directed to look at the reflection of his eye in the mirror.

Interpretation of radiographs.—The diameter of the silver ring is known and its measurement is checked on the X-ray film. Any radiographic magnification is noted. This has never been more than 1 mm., and when present allowance must be made for this fact. When an anode film distance of 30 inches or more is used parallax magnification of the ring is negligible, and a schematic eye of 24 mm. drawn on the film is accurate enough. In the lateral view a line is drawn posteriorly from the centre of the ring and at right angles to it for about 22 mm. The upper and lower limit of the ring is used in turn as the centre of a circle whose radius is 12 mm. (i.e., half the average length of an eye 24 mm.); with a pair of dividers set at 12 mm. the horizontal line is intersected by arcs described from the above centres. The point of intersection on the horizontal line is now taken as the centre of a circle with 12 mm. radius and this is described on the radiograph. A similar procedure may be done with the silver ring in the second position, that is, looking down. Likewise in the postero-anterior view a circle of 12 mm. radius is described around the centre of the silver ring. The ring is known to be at the limbus and so measurements may be taken from this on both the postero-anterior and the lateral radiographs.

Movement of the foreign body is the essential diagnostic feature. If the relationship of the silver ring image to the foreign body is unaltered in the first and second positions it may be presumed that the foreign body is moving with the eye. The radiographs with

the schematic eye marked upon them will indicate also the position of the object with regard to the centre of rotation. If the foreign body is in front of this it will apparently move with the ring, if behind against it—that is, a foreign body in the posterior half of the eye will lie at a higher level in the second position where the ring has rotated downwards.

The shadow of the foreign body might fall within the circles described and yet be outside the eye. The point is settled by the movement of the foreign body, which is generally absent when it is extra-ocular. There is, however, an exception to this in the case of a foreign body in Tenon's capsule, where slight movement may take place, but this is rarely as great as in the case of an intra-ocular foreign body. In such cases, therefore, if the foreign body shows a new relationship to the ring in the second position it is probably outside the eye. When the foreign body is clearly outside the limits of the circles described then it is certainly extra-ocular. The size of the foreign body is carefully measured on the radiograph. This will determine the length of the scleral incision necessary for its extraction.

Ahlbom's method is also good and simple. A circle of 26 mm. diameter is placed in front of and projected on to the eye. He uses teleradiography. The distance between the anti-cathode and the film is 2.75 m. and the projection is practically cylindrical and the error in magnification so slight that it can be neglected. To show very small foreign bodies he uses an aluminium disc between the occiput and the cassette and also employs the "bone free" technique.

(5) "*Bone-free*" (Sans squelette).—The osseous shadows of the orbital walls and floor of the skull sometimes obscure the presence of small intra-ocular foreign bodies particularly fragments of 0.5 mm., and even 1 mm. when the material of the foreign body is of a kind which does not give a dense shadow.

"Bone-free" exposures of the eye anterior to the equator may be obtained by turning the head partly to the side away from the affected eye and placing the X-ray tube at a tangent to the lateral orbital margin and the film at the inner canthus.

This procedure although helpful in difficult cases has its limitations.

(6) *Injectations into Tenon's capsule*.—Efforts have been made to outline the sclera by the injections of radio-opaque substances into Tenon's capsule.

Air has been used. In some cases it remains for a day or two and has had to be sucked out. There is an instance recorded in the literature of the death of a rabbit from air embolism after injection into Tenon's capsule. Lipiodol has been used, and in the United States, diotrast.

Pirie has described the " subjective " method of localisation in a dark-adapted patient, the retina being stimulated by X-rays the shadow of the foreign body is projected. The fallacies of this procedure are that a retinal injury, such as the site of ricochet of the foreign body will produce a shadow and the shadow of the foreign body may fall upon the blind spot.

Biplane fluoroscopy is difficult and liable to inaccuracies and Thorpe's endoscope is a traumatic approach to localisation of an intra-ocular foreign body which is not justifiable in most cases.

Locators

In 1851 Aveling used a magnetised needle for the detection of the site of a steel foreign body such as a needle embedded in the soft parts of the body. The needle dipped and adhered to the skin at the exact point under which the foreign body lay.

Pooley and Pagenstecher in 1880 and 1881 working independently devised an apparatus also based on the principles of magnetic localisation. Their experiments showed that the depth of the foreign body might be inferred from the degree of deflection of the magnetised needle and a change in the position of the foreign body after the application of the magnet may be ascertained.

In 1894 Asmus added a mirror to such an apparatus to reflect the excursions of the magnetic needle on to a scale. Berman's locator described by Minsky (1944) is a rod in which are placed the equivalent of two transformers, one in the handle and the other at the tip, which is used to search for the foreign body. The primary coils are connected in series to a source of alternating current. Also in series, the secondary coils are connected through an amplifying unit to a voltmeter. When an alternating current is sent through the primary coils, a current is produced in the secondary coils by induction. The instrument has a means of equalising (balancing out) the voltages in the secondary coils so that the needle of the voltmeter will read approximately zero, since no current flows between them. If the coil in the tip of the rod approaches a magnetic metal (the foreign body), the balanced inductance is disturbed and a difference in potential takes place in the secondary circuit, which results in a flow of current. The amount of this current, shown by the deflection of the needle in the voltmeter, varies with the size of the metallic particle and with its distance from the tip. At the greatest point of deflection, therefore, the tip of the locator is immediately over the foreign body. Conversely, as the locator travels away from the foreign body, the deflection of the needle is lessened. Minsky states that he can estimate the depth of a foreign body, if its size and composition are known, by determining the distance necessary to give the

same reading, with the controls unchanged, in approaching a similar piece of metal. The instrument responds best to iron and steel, and less effectively to copper, brass, silver, aluminium, lead and their combinations. The differentiation of a non-magnetic from a magnetic foreign body is easily made when the needle of the voltmeter does not move at all.

Magnets

The best magnet is, I think, one shaped like a 6-inch shell with screw-in straight terminals; some are short and conical and others long and attenuated and about 2 to 3 mm. in diameter at the tip, the latter are more accurate and easily manipulated than the former which give a more diffuse field and may attract the foreign body to the side of the cone. This magnet is suspended over the head of the operating table. It should be so perfectly counter-balanced that the slightest move or tilt leaves it in place. The magnet itself should be so fitted on a ball-bearing setting that it may be tilted and turned easily to any angle, and fixed so by a clamp. A foot-switch is controlled by the surgeon to make and break the electric current. The patient's head is immobilized between sandbags and covered with a dark green linen mask with an aperture for the eye to be operated on. The main body of the magnet is enclosed in a sterile dark green linen sleeve. The magnet terminals are sterilized in A.C.10, rinsed in saline, dried with a sterile cloth before being screwed into the socket at the end of the magnet.

The advantage of this magnet is that the terminal tip may be applied with greater precision and kept so as long as necessary. Also it permits ophthalmoscopic examination of a vitreous foreign body by the surgeon to ascertain the magnetic nature or not of the foreign body.

Such a magnet is obviously desirable in military surgery where multiple wounds are present and on account of these the patient must lie on the table. It is even so in civil injuries when the eye alone is affected.

Solus Magnet. This has been used in military surgery, particularly in eye units operating in the field. It was intended for use as a portable magnet, and supplied with a strap for slinging over the shoulder of a medical orderly. The orderly has great difficulty in maintaining the end of the magnet at the desired site, and this procedure is technically unsatisfactory. Lt.-Colonel A. Lister effected a considerable improvement in this respect by mounting the magnet on a portable X-ray stand (Fig. 1).

On the upright of the stand there is a ratchet adjustment to lower the tip of the magnet to the eye. As the movement of this is somewhat coarse I found it best to stop when the tip of the terminal was

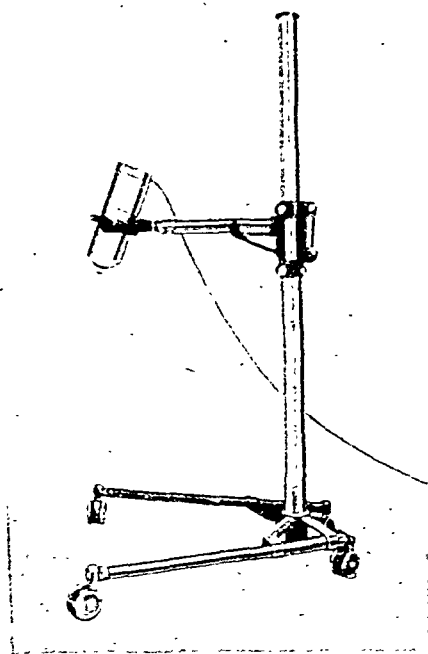


FIG. 1.

Solus giant electro-magnet mounted on portable X-ray stand.
(Reprinted from Eye Surgery, H. B. Stallard, John Wright and Sons, Ltd. 1946).

2 or 3 mm. from the cornea or sclera and then by gradually raising the operating table the magnet terminal could be brought into contact with the cornea or into the lips of a scleral wound.

An improvement could be effected in this magnet by making the collar and framework holding the magnet of non-magnetisable metal and providing a clamping device to keep the magnet fixed for, when placed in an oblique position, it kicked.

Fig. 2 shows another type of counter-balanced magnet set in a hinged frame. The magnet is swung over the head of the operating table and held inclined at an appropriate angle.

The force inside the ring of Mellinger's magnet—that is, the operative field—is homogeneous all over. There is no preferred point of force and no dispersion of lines of force in the operative zone. It is possible to make with Mellinger's magnet terminals more delicate manipulations than with Haab's magnet.

The terminal held in the surgeon's hand must never be introduced into the operative field without the current having been switched on. The danger of placing the terminal inside the ring and then turning on the current is that it may become dragged suddenly on to the eye.

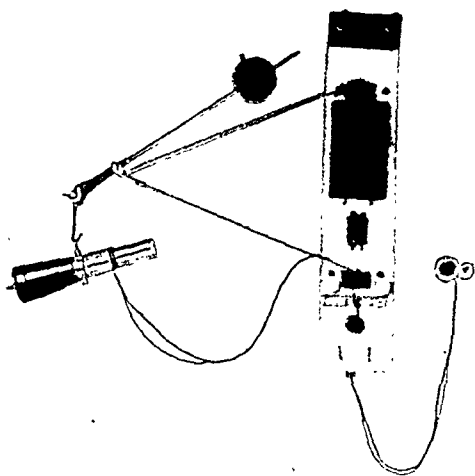


FIG. 2.

Counter-balanced giant electro-magnet. (*Reprinted from Eye Surgery, H. B. Stallard, John Wright and Sons. 1946*).

Mellinger's magnet has several disadvantages. The patient complains of an oppressive and confined feeling when his head is within the ring. The surgeon has often to exert considerable manual strength to hold still the magnet terminal in the magnetic field. If this is protracted it may impair slightly the steadiness of his fingers for subsequent stages in the operation. It is sometimes difficult to tie the scleral suture with the head inside the ring after extraction of a foreign body by the scleral route. In such cases the ring has to be swung clear of the head before this stitch can be tied, thus causing a short delay in closing the wound.

It is most important not to assume that because the application of the magnet causes no pain in the eye the foreign body is therefore non-magnetic. Had I believed this I would have lost 20 foreign bodies in the M.E.F. series of 102 cases and 19 in the B.L.A. series of 72. All gave rise to no pain on testing, and all (39) were extracted by the giant magnet, 36 by the scleral route and 3 by the anterior route.

The low magnetic quality of certain foreign bodies will be increased when placed in the field of a giant electro-magnet, but only to a certain extent, saturation being reached very quickly. For this reason many applications of the magnet are unnecessary for when this is done heat is generated in the wiring and the pulling power of the magnet becomes less. I have extracted a small magnetic foreign body on the fourth application of the magnet.

Kraus and Briggs recommend up to 50 applications for 2 seconds duration at 2 seconds interval. They comment that if it is not extracted with 50 applications it is useless to go on.

In one case in the B.L.A. series—Pt. J.—a metallic foreign body 10 x 4 x 3 mm. entered the sclera in the upper temporal quadrant of the right eye in front of the equator. The foreign body traversed the vitreous and part of it had perforated the sclera about the equator and beneath the internal rectus muscle. When the magnet terminal was placed over the wound on the temporal side the eyeball rotated temporally. The scleral wound on the temporal side was closed by sutures and covered with a conjunctival flap. The internal rectus muscle was then exposed, mattress sutures inserted in the muscle belly and its tendon divided. Diathermy was applied around the scleral wound through which part of the foreign body was projecting. Three scleral sutures were inserted on either side of the projecting part of the foreign body, the scleral wound was enlarged and the foreign body extracted by the magnet.

In one case of traumatic cataract when a magnetic foreign body 0.25 mm. was brought forward by the anterior route it became arrested in a fold of the torn lens capsule and would move no further. It was extracted by Arruga's forceps passed through a keratome section.

Surgical exploration

In some cases where it is doubtful from the radiographs whether the foreign body is just inside the eye, impacted in the sclera or in Tenon's capsule surgical exploration is indicated. The diathermy apparatus and the instruments necessary for incising the sclera and extracting the foreign body by a giant electro-magnet are available.

A tongue-shaped flap of conjunctiva and Tenon's capsule convex towards the limbus is cut and reflected posteriorly on its base, the axis of the flap being in line with the site of the foreign body and its apex 7 mm. or more in front of the site of the foreign body. The flap is held reflected by two sutures of 00 black silk clamped to the head towels.

Tenon's capsule is dissected from the sclera with a few strokes of a small muslin swab held in forceps. Adhesions of Tenon's capsule to the sclera, bands and nodules of fibrous tissue are guides to the site of the foreign body. The sclera is dimpled where it is cut tangentially or penetrated by the foreign body. The foreign body may be found inside a fibrous nodule on the sclera or it may be partly embedded and transfixing this structure. If the foreign body has passed into the eye the entry wound is circumvallated with diathermy and the sclera opened between sutures taking

care not to press the foreign body into the vitreous as it lies in the choroid or deeper layers of the sclera. Extraction is effected either by forceps or the electro-magnet. After closure of the scleral wound penicillin powder is lightly dusted in its vicinity and the conjunctival flap sutured with a continuous key pattern stitch.

Operation

The scleral (posterior) route was used in 45 cases in this series. In 37 of these the foreign body was extracted, in one it was removed with special forceps, and in the remaining 7 the foreign body was not attracted to the tip of the magnet. I have a feeling that in some of these with a better magnet and more accurate X-ray localisation the foreign body might have been extracted. Most of the cases in which the foreign body was not extracted occurred at a time when conditions were particularly crude and the field electrical generating set full of vagaries and inconsistent in its behaviour. The technique of the posterior route is fully described in the account of the M.E.F. series of 102 cases (*Brit. Jl. Ophthalm.*, Vol. XXVIII, p. 105, 1944).

Technical essentials are a tongue-shaped conjunctival flap convex anteriorly, the application of diathermy to the site on the sclera nearest to the foreign body and over an area of sufficient length to cover an antero-posterior linear incision 1 mm. longer than the greatest diameter of the foreign body, the insertion of a mattress scleral suture on either side of the scleral incision when about two-thirds of the thickness of the sclera had been cut through, and turning the head and operated eye so that the scleral wound lies uppermost before incising the choroid. The latter point is important in the prevention of vitreous presentation in the scleral wound. In no case was any vitreous lost. Nor did the diathermized area of the choroid and retina embarrass the extraction of a foreign body. Some surgeons believe that the foreign body may become entangled in the coagulum and so circumvallate the scleral incision with diathermy, leaving the area of incision untreated.

It was necessary to introduce the tip of the giant electro-magnet terminal into the lips of the wound so that its point touched the face of the vitreous but did not enter it. This was done by fine adjustment in the height of the magnet terminal.

The scleral suture was tied immediately after extraction of the foreign body and penicillin powder was lightly dusted into the wound.

Fifteen foreign bodies were extracted by the anterior route, 13 of them by the magnet and 2 by forceps.

In 12 instances there was no movement of the foreign body on applying the giant electro-magnet.

In some cases the foreign bodies were 0.5 mm. in size and impacted in the lens where they had produced a localised opacity near the equator and no appreciable visual disturbance. In these it was probable that they were magnetic for similar fragments removed from the face were so. It was decided to leave the fragments alone and to review the eye from time to time. It was undesirable to draw these through the lens capsule, and in any case it was improbable whether a magnet terminal placed on the cornea would have moved such minute particles of an alloy.

I do not believe that the bogey of retinal detachment after the scleral route extraction is any more probable and indeed less so than the risk of vitreous loss which is also quoted as a contra-indication to the scleral route method of extracting intra-ocular foreign bodies. With careful technique neither of these serious events is likely to happen.

If a retinal detachment occurs as a sequel to a foreign body it is due to the nature of the penetrating wound and the pathological changes this has caused inside the eye rather than any operative intervention of a careful and properly planned character.

Cases

Table D gives the units to which the wounded and injured men belonged. As might be expected the infantry and armoured troops were prominent in the battle casualties and R.E.M.E. and R.A.S.C. in accidental injuries in field workshops.

Table E shows the type of missile which penetrated and was inside the eye in the two groups of casualties (1) battle and (2) accidental.

It will be seen from this table that as in the M.E.F. series shell fragments accounted for the largest number of eye wounds with retained intra-ocular foreign body. Land-mine injuries were next common whereas in the M.E.F. series these were third in the order of frequency, hand grenade wounds being second. In Normandy the orchards and verges of the roads were heavily mined. The "shoe" mine was particularly difficult to detect. The charge was encased in a wooden box, a trip wire projected above the surface of the ground and was difficult to see. Soldiers walking or running over such mines often had a foot blown off, the line of traumatic amputation being at the ankle, and had an injury to an eye. In the case of one officer a fragment of the rubber boot which covered his amputated foot was blown into his right orbit and lay on the sclera, producing a severe concussion of the intra-ocular membranes on the temporal side.

There was a higher incidence of eye injuries from fragments of machine-gun bullet casing than in the M.E.F. series. This may

have been due to the severe fighting at close quarters in villages (Tilley, Villers-Bocage, Hottot, Caumont and others) where machine and Sten gun fire was used liberally in house to house attacks, fragments of bullet casing produced by ricochets spattered the exposed faces of the combatants.

TABLE D

Unit	Battle Casualty	Accident	Total
British Infantry ...	21	1	22
Canadian Infantry ...	5	—	5
R.A.C. ...	7	2	9
Canadian Armour ...	1	1	2
R.A. ...	3	1	4
R.E. ...	3	1	4
R.E.M.E. ...	3	2	5
R.A.S.C. ...	1	3	4
Royal Marines ...	1	—	1
R.A.F. ...	—	1	1
U.S. Engineers ...	1	—	1
Polish Infantry ...	5	—	5
Polish Armour ...	1	—	1
Polish Medical Corps	1	—	1
Dutch Infantry ...	1	—	1
Yugo-Slav Infantry...	1	—	1
German Infantry ..	4	—	4
German Navy ...	1	—	1
Total ...	60	12	72

TABLE E

Missile	Battle Casualty	Accident	Total
Shell	17		17
Hand Grenade	4		4
Machine gun bullet casing	8	1	9
Land mine	14		14
Booby trap	4		4
Mortar bomb	6		6
Bazooka bomb	3		3
Aerial bomb	2		2
Phosphorus bomb	1		1
Hammer and chisel		8	8
Tank track	1	2	3
Glass		1	1
Total	60	12	72

In the attack on Caen the German mortar fire was exceedingly heavy and so were the British casualties. Booby-traps were left in the breaches of abandoned German weapons and in vehicles and houses. The "bazooka" bomb was used against the armoured units and casualties from this missile also occurred in the infantry and engineers.

In the Western Desert Campaign we believed that an improved steel helmet of the German coal-scuttle pattern with side-pieces protecting the temporal region on each side would have saved a number of penetrating wounds of the sclera. There is no doubt that the number of serious head injuries would have been appreciably reduced. N.C.O.'s ordered the men to tighten the lace inside the British steel hat. This procedure raised it an inch or more on the head and so increased the exposure of the temporal fossa and the lateral side of the orbits.

As in the Western Desert engineers clearing mine-fields did not or would not wear a protective device over their eyes. Many severe injuries of both eyes from blast, burning and the spatter of gun-powder, earth and stone fragments could have been prevented by wearing thick salvoc goggles. Later in the campaign the eye pieces of captured German respirators were used to good prophylactic advantage.

The hammer and chisel provided the highest incidence of accidental penetrating wounds with retention of an intra-ocular foreign body.

Evacuation of wounded

In Normandy the tented field hospitals were about 8 miles behind the line during the battles for Caen and Caumont. We operated most of the time as a Casualty Clearing Station. The wounded came to us from the field and generally reached us within a few hours, or at the most within a day, of being hit. We could not hold them for more than a few days and as soon as they were fit to travel they were evacuated either by air or sea to the United Kingdom. At times it was possible to keep some patients until the injured eye was out of immediate danger. At first the conditions of work were crude, operating tables stood at odd angles on an uneven field, water was limited to one enamel bowl shared by four surgeons and changed once every two hours, gloves and gowns were reserved for intra-abdominal work, fitful illumination depended on the vagaries of a field generating set, primus stove sterilizers had irregular habits and unseasoned theatre orderlies slowed down the rate of dealing with a large number of casualties. It was surprising how quickly matters improved, and efficiency and good order became established.

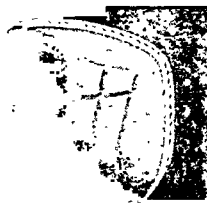
I do not think that any eye casualty suffered from an operation under these conditions and during the 10 weeks we worked thus all the eyes which received conservative surgical attention settled down well after operation, none became infected and in none did removal of the eye seem likely to be necessary at a later date. This was just the fortune of war.

After the break-through at Falaise and the advance across the Seine, Picardy and Flanders I was transferred to the 108 (General) Hospital (2,000 beds) which was working on the outskirts of Brussels in a bottle-neck on the line of evacuation of casualties. Here in a separate pavilion there were 90 ophthalmic beds, treatment rooms and operating theatre suite. The volume of work was immense and until the lull that followed von Rundstedt's advance in the Ardennes we operated as a C.C.S. I was, however, allowed at times to hold seriously wounded eye cases for 2 or 3 weeks and

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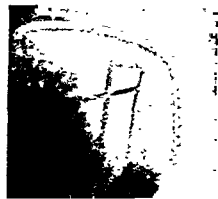
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this was so after the Rhine crossing and until the end of the war in Europe.

In May, June and July, 1945, repatriated prisoners of war of many European nationalities, "displaced persons" and enemy prisoners required surgical attention for neglected wounds and were held until fit for discharge from hospital.

Clinical Facts

An enquiry was made about the distance of the explosion from the soldier. Whilst the majority of wounded were able to assess this some were unable to appreciate which of many missiles bursting near them caused the wound.

One man was wounded by an aerial bomb explosion 200 yards away whilst he was lying down and in 7 per cent. of shell wounds this missile burst more than 20 yards from the soldier. The majority were within this range.

Fifty per cent. of grenades and 34 per cent. of mines and booby traps exploded within a yard of the soldier.

The men were asked their position when hit and the direction from which the missile came. In some, but not all cases, such information was helpful.

Size of the missiles.

Maximum diameter less than 1 mm.	10
1 to 2 mm.	16
2 to 3 mm.	17
3 to 4 mm.	7
4 to 5 mm.	6
5 to 6 mm.	3
6 to 7 mm.	3
7 to 8 mm.	1
10 mm.	1
11 mm.	1

There were 6 cases of multiple foreign bodies in the eye and in 3 of these all the foreign bodies were removed by a single operation through the scleral (posterior) route. In the fourth case the foreign bodies were not magnetic. In one case there were two foreign bodies in the vitreous measuring $4 \times 2.5 \times 1$ mm. and $4 \times 1 \times 1$ mm. respectively; in another case 3 foreign bodies all measuring 0.5×0.5 mm., the third case 2 foreign bodies 1×1 mm. and 1×1 mm., the fourth 2 foreign bodies 5×4 mm. and 2.5×1 mm., the fifth 2 foreign bodies 3×0.5 and 0.25×0.25 mm. and the sixth 2 foreign bodies less than 0.25 mm. both situated in the lens cortex.

As in the M.E.F. series foreign bodies between 2 to 3 mm. in their maximum diameter were common and the majority were under 3 mm.

Age incidence.

18 to 19 years	3
20 to 30 years	57
31 to 40 years	8
over 40 years	4

Eye affected.

Right eye	35
Left eye	27
Both eyes	10

Site of penetrating wound.

(1) Through the cornea.

Upper temporal quadrant	5
Upper nasal quadrant	6
Mid-line between these quadrants	3
Centre of cornea	2
Corneo-scleral junction on nasal side	1
Lower nasal quadrant	6
Lower temporal quadrant	11
Mid-line between these quadrants	2

(2) Through the sclera.

Upper temporal quadrant	10
Upper nasal quadrant	4
Mid-line between upper and lower nasal quadrants	1
Lower nasal quadrant	6
Lower temporal quadrant	5

(3) Multiple—2. In one of these two penetrating wounds were in the lower nasal quadrant of the sclera and in the other there were three wounds in the cornea, 2 in the lower nasal and 1 in the upper temporal quadrant of this structure.

(4) Wound of entry not seen—9. In the majority of these exploration showed that the foreign body had entered the orbit and penetrated the sclera behind the reflection of the bulbar conjunctiva.

In this, the B.L.A. series it is evident that the site of the penetrating wound was commoner on the temporal side than the nasal, both in the cornea and the sclera, whereas in the M.E.F. series there was a high incidence of wounds in the nasal half of the cornea, particularly the lower nasal quadrant, and in the lower quadrants of the sclera.

Uveal Prolapse—7 cases. The nature and size of the missiles which caused the penetrating wound and prolapse is set out below.

Iris.

Shell	3 x 1.25 x 0.75 mm.	1
	5 x 4.5 x 1.5 mm.	1
	2 x 1.25 x 0.25 mm.	1
	3 x 2 x 0.25 mm.	1

Iris and Ciliary Body.

Phosphorus bomb	6 x 4 x 0.5 mm.	1
Land-mine	10 x 4 x 3 mm.	1

Ciliary Body.

Bullet casing	2 fragments 5 x 4 and 2.5 x 1 mm.	1
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Choroid.

Aerial bomb	2.5 x 1.75 mm.	1
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The two eyes in which there was prolapse of the ciliary body from a large fragment which traversed the vitreous and stuck partly through the sclera on the opposite side of the globe became quiescent for a few weeks but ultimately required excision. The others were saved.

Traumatic cataract.—34 cases. In 24 of the 34 cases the penetrating wound was in the cornea. In 7 of these the damage to the lens was slight. The size of the intra-ocular foreign body in these cases varied from 0.12 to 1.5 x 1 x 0.25 mm. In one instance it was 0.12 mm. and in another there were 2 fragments of aluminium alloy about 0.25 x 0.25 mm. deep in the lens.

The ultimate visual result in these cases was on the whole good. Two had 6/6 vision, one 6/9, another 6/24 before leaving hospital and no information about the others was forthcoming from the hospitals to which they were evacuated in the United Kingdom.

The remaining 17 cases had severe damage to the lens.

In 6 instances the lens was damaged where the foreign body passed through the sclera. Two of these showed a slight degree of cataract and in 4 it was severe.

The wound was not seen in 3 cases of traumatic cataract and cataract was bilateral in one case, one eye had a scleral wound and in the other eye the wound was not seen.

Vitreous haemorrhage.—The incidence of vitreous haemorrhage in the B.L.A. series was 44.4 per cent., that in the M.E.F. 48 per cent. and the proportion of slight and severe cases was nearly alike in the two series, the severe cases being slightly more in the B.L.A. casualties. Table H shows the incidence of slight and severe vitreous haemorrhage in relation to a corneal or scleral penetrating wound.

TABLE H

Vitreous Haemorrhage	Corneal wound	Scleral wound	Wound not seen	Total
Slight ...	4	3	2	9
Severe ...	3	20		23
Total ...	7	23	2	32

As might be expected severe vitreous haemorrhage occurred when the site of the entry wound was near the exit of a vena vorticiosa, in the line of a long ciliary artery and in some cases when it traversed obliquely the pars plicata of the ciliary body. In such cases the sizes of the foreign bodies were from 2.5 to 6 mm. in their longest diameter.

Vitreous prolapse.

On arrival at hospital vitreous prolapse was present in 5 cases. In 4 of these it was through a scleral wound inflicted by foreign bodies 1, 2.5, 3 and 5 mm. in their longest diameter and in 2 of these cases the foreign body had also perforated the upper lid. In one case vitreous prolapsed through a penetrating wound of the cornea made by a 2 mm. foreign body.

Retinal detachment.

Retinal detachment was present in 2 cases. In both of these the entry wound was in the lower half of the sclera, in front of the equator and the retinal detachment and vitreous haemorrhage were in the vicinity of the wound.

In two instances when the vitreous haemorrhage was too severe to see the retina, some dark brown blood-stained inter-retinal fluid escaped when the choroid was incised for magnet extraction of the foreign body. So it is probable that retinal detachment was present in these cases.

I have no knowledge of the incidence of late retinal detachment in this series for it has been impossible after the war to trace more than a small number of them.

(Follow-up cards were sent with the notes of every soldier in this series. Out of 72 I received only 1 reply, and that from a general house-surgeon in a small north country hospital, and this contained no details. This was a similar experience to the Middle East series.)

Visual result.

It was impossible in this series to obtain facts about the ultimate visual result. During 1944 we operated as a casualty clearing station and as soon as a man was fit to be moved he was evacuated to the United Kingdom. As stated above neither follow-up cards nor letters about the wounded were answered.

Whenever it was possible for a patient to have his vision tested before evacuation this was done (in the Normandy campaign this examination was made in a tent). It is possible that in many cases the visual acuity improved later. Such records as were taken at the time of evacuation are as follows:—

	6/5	2 cases
	6/6	5 cases
	6/9	5 cases
	6/18	1 case
	6/24	1 case
	6/36	2 cases
	Counting fingers			...	2 cases
	Hand movements			...	8 cases
Perception of light.	{	Accurate projection			2 cases
		Inaccurate projection			5 cases
No perception of light.	{	2 cases

In the remaining 37 cases visual acuity was not tested because of evacuation at short notice and the impossibility of doing an accurate refraction on a patient with multiple wounds lying on a stretcher.

Excision.—Two eyes were ultimately excised. In each of these the foreign body had passed through the limbus of the upper nasal quadrant, traversed the vitreous and was impacted in the sclera of the lower nasal quadrant, the foreign body being partly inside the eye and part of it projecting through Tenon's capsule. In each case the foreign body was large, in one case it was 10 x 4 x 3 mm. and in the other 5 x 4.5 x 1 mm. In both cases after wound toilet, extraction of the foreign body and suture of the scleral wounds the eye settled down and remained so for a few weeks. I heard indirectly that the ophthalmologist in the Canadian Hospital to which these men were transferred considered excision proper on account of no perception of light, a soft eye and a wound in the ciliary region.

Endophthalmitis.—There were 2 cases of endophthalmitis which settled down. These received parenteral and subconjunctival penicillin.

There were no cases of panophthalmitis. How much this was due to penicillin powder dusted on to the exposed sclera around

the sclerotomy wound in the scleral route extractions it is impossible to say. The surgical team wore gloves and a non-touch technique was adopted. The post-operative course was uncomplicated by inflammation in all cases of scleral route extractions.

Associated with wounds in other parts of the body.—Wounds were present in other parts of the body in 51.4 per cent. in the B.L.A. cases. Multiple wounds included severe injuries such as open fractures of the femur, haemothorax, amputations, burns and serious head injuries. The nature of these is recorded briefly in the appendix.

The variety of multiple injuries due to hand grenades, land mine explosions, and booby traps conformed closely in both the M.E.F. and B.L.A. series.

Incidence of penetrating wounds of the eye with retained intra-ocular foreign body

In both the M.E.F. and B.L.A. series it has been difficult to obtain accurate statistics of the incidence of penetrating wounds of the eyes with retained intra-ocular foreign bodies in relation to eye casualties and to all battle casualties. Security measures often prevented a publication of numbers of casualties particularly in the M.E.F. When the German-Italian forces reached Alamein in June, 1942, many records at H.Q. Medical Services were destroyed. The transfer of wounded through several hospitals and the collection of cases requiring magnet work at three of these (the majority of such cases went to the 15th Scottish and the 8th General Hospitals and a few to the 6th General Hospital) made the keeping of accurate statistics impossible.

In the M.E.F. series there were 102 cases of a penetrating wound of the eye with retained intra-ocular foreign body out of 328 men with wounds within the circumference of the orbit and 110 with intra-cranial wounds affecting the visual pathways. In some of the latter the orbit and the eye were also injured.

Scott and Michaelson collected statistics of the terminal phase of the Western Desert campaign and found 78 cases of intra-ocular foreign bodies out of 301 eye battle casualties, 190 of which had penetrating wounds of the eyes. In 58 of this series both eyes were injured.

Dansey-Browning in the Italian campaign had 24 cases of intra-ocular foreign body out of 129 eye casualties in whom 148 eyes were injured. He considers that eye casualties were 2.5 per cent. of the total battle casualties.

In this B.L.A. series there were 72 cases of retained intra-ocular foreign body in 335 battle casualties and 62 accidental injuries of the eyes.

The statistics given by Lt.-Colonel A. Lister in his Summary of Ophthalmic work in 21 Army Group from June 5 to September 30, 1944, are 2,188 eye casualties of which 1,440 were due to direct enemy action, 480 (about 30 per cent. of battle casualties) had a penetrating eye wound, 157 had intra-ocular foreign bodies, 51 (about 30 per cent.) were removed.

From October to December, 1944, eye casualties due to direct enemy action were 595, and were 3.8 per cent. of all casualties in October, 3.1 per cent. in November, and 0.8 per cent. in December. There were 215 "battle accident" casualties in this same period. Twenty per cent. of the eye casualties had penetrating wounds of the eye and 33 per cent. of these had intra-ocular foreign bodies, only 25 per cent. of which were removed.

Whiting and Goulden comment that in July, 1916 (during the 1914-18 war) they had 30 cases of retained intra-ocular foreign body in this month, 5 cases occurring on one day and 13 in a week. They also remark that in France at this time a considerable number of intra-ocular foreign bodies were non-magnetic.

Cridland gives the incidence of penetrating wounds of the eye with retained intra-ocular foreign body in civil practice in 3 large industrial areas as 1 in 977.9 of all eye cases and 1 in 299 of accident cases.

Summary

The clinical facts of 72 cases of penetrating wounds of the eye with retained intra-ocular foreign body in the B.L.A. are surveyed. Methods of X-ray localisation and types of giant electro-magnet are discussed. In military surgery under field conditions such apparatus has to be simple for the former has to be used expeditely by men with no special knowledge or training. There is indeed little time for such work when dealing with almost daily convoys of several hundred wounded. Our average time for a case was three minutes for suturing a limbal ring in place, about 10 minutes for the X-ray and 7 to 12 minutes for the operation of extraction of the foreign body by the scleral route.

The scleral route for extraction is indicated in the majority of cases and is simpler and less traumatic than the anterior method. With careful technique the danger of vitreous loss is negligible. This complication did not occur in this series. Retinal detachment is mentioned in the literature as a complication of the scleral route operation. I think this catastrophe is unlikely to occur as a direct result of the operation when diathermy has been used around the incision in the sclera and intra-ocular membranes. It may have happened in cases where diathermy was not used but there is no comment on this point in the literature. Retinal detachment is

more likely to be due to the pathological changes induced inside the eye at the time of injury.

In this series 69.3 per cent. of foreign bodies were extracted, 51.3 per cent. by the scleral route and 18 per cent. by the anterior route. Other authors including 21 Army Group Medical H.Q. state that about 30 per cent. of modern war missiles are extracted by a giant electro-magnet but they do not differentiate the incidence of success by either the anterior or posterior route. Many reported failures by the anterior route might not have been so if the scleral route had been properly tried.

With better X-ray facilities for localisation and a better magnet I feel that the figure of successes in this B.L.A. series might have been higher. For extraction of the feebly magnetic alloys of war missiles it is essential to bring the terminal of a very powerful electro-magnet into the lips of a scleral incision placed as near as possible to the foreign body.

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"BLUE HALOES" IN ATEBRIN WORKERS

BY

IDA MANN

OXFORD

I AM deeply indebted to Dr. H. Wyers for having called my attention to this condition and for the opportunity of examining the cases described.

Since the introduction of atebtrin* for the treatment of malaria and its consequent manufacture on a large scale, certain pathological conditions consequent on handling it in bulk have become known.

* Atebrin is 2-chloro-5-(ω -diethylamino- α -methylbutylamino)-7-methoxyacridine dihydrochloride and is also known as Mepacrin (B.P.) and Quinacrin (U.S.P.).

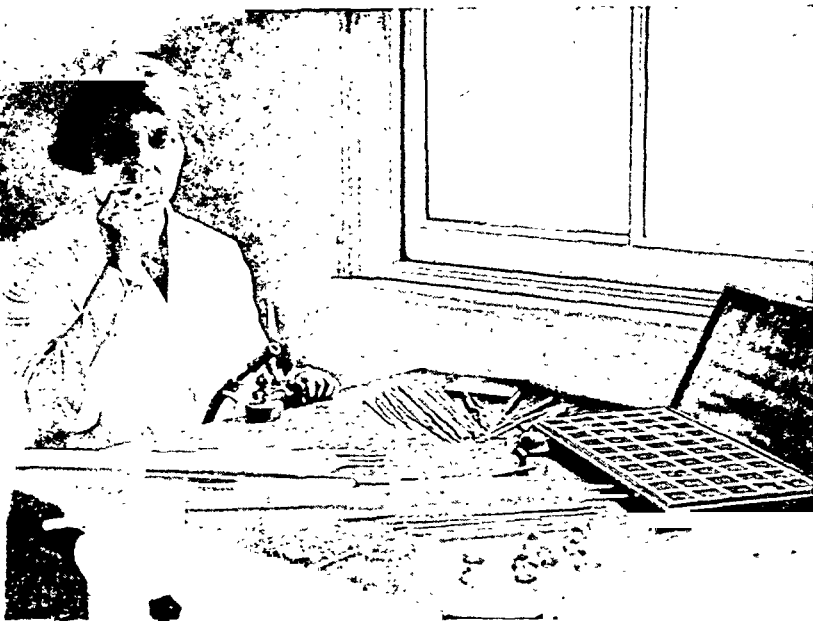
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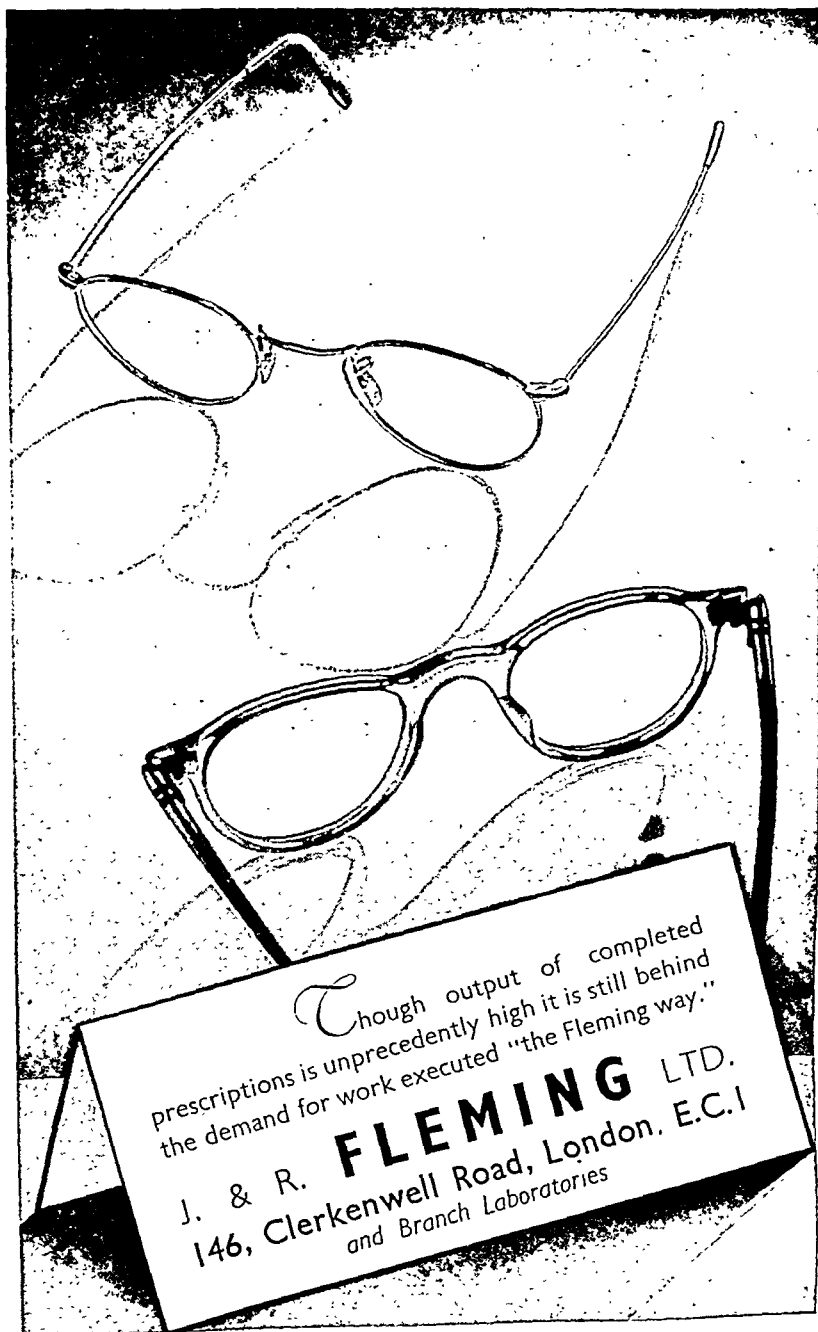


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The commonest appear to be erythematous and papular skin eruptions on exposed parts, with puffiness of the eyelids, lacrimation and pain over the frontal sinuses. These symptoms occur in certain sensitive individuals who are usually rendered thereby unsuitable for work with the drug and are transferred to other processes. Among those workers, however, who do not show this special sensitivity and therefore continue in the work, a yellow discoloration of the skin and conjunctiva and a curious corneal condition may develop. The present note concerns the eye condition of six workers engaged in the production of the drug and its compression into tablets. This eye condition is not a scheduled industrial disease and so is not compensatable in the ordinary way.

Ocular signs and symptoms.—The patients, healthy men aged 28, 35, 36, 49, 53 and 66 respectively, had been working in an atmosphere which often contained for short periods clouds of very fine atebirin dust. One had worked in the plant for a year, two others for 10 months. All considered that they were not particularly sensitive, and experienced no dermatitis or other discomfort. They wore protective clothing, goggles and masks, but the dust was so fine that in spite of this they soon noticed yellow staining of the conjunctivae and skin, especially of hands, head and neck. The men worked 9 hours a day for five days a week and after some weeks or months they all noticed that when looking at a small source of light at night they saw a blue halo round it. There was no ocular pain, no lacrimation or photophobia. All insisted that the halo was mainly blue, that for a light one metre away it began approximately two inches from the light and was about three inches wide. It was darker blue near the light and pale blue at its outer edge. If the light was placed at six metres, faint yellow, green and reddish brown bands appeared outside the blue ring. These were usually not noticed until asked for, the blue ring being much the brightest. All the men stated that their visual acuity was unimpaired and on examination it was found to be 6/6 in all the eyes. They could see the blue halo at any time by looking at a light and there was never any pain. Two men stated in addition that their nostrils were sore and occasionally bled.

Slit-lamp appearance.—On examination the condition in all six patients was similar. In some it was more severe than in others.

In the fully developed condition there was a yellow discoloration of the conjunctiva in the interpalpebral space (Fig. 1) and in addition a curious corneal condition, just visible macroscopically as a slight dulling and yellowing of the cornea. A slit-lamp, however, resolved these appearances as follows :—

1. The conjunctiva showed a diffuse pale yellow stain in the interpalpebral space only. The vessels were not engorged nor was there any increase in secretion. At the limbus in the exposed portion and also just under the edge of the lower lid, but not at the upper quarter of the limbus, there were aggregations of minute dark brown dots. These are seen in Figs. 2 and 3. They resembled very much the pigment dots seen here in Chinese and other lightly pigmented races and in some Southern Europeans, but they were rather darker, denser and more finely particulate than these. They appeared to be an actual deposit, possibly picked up by cells (macrophages) and lying in their cytoplasm or possibly merely existing as a fine surface dust. They were not movable and could not be washed off.

2. The cornea showed a remarkable change. The whole surface, even that covered by the upper lid, was peppered with very fine dust-like particles. These appeared dark yellowish brown by direct illumination, and quite opaque by transmitted light. The size of the particles varied but at a rough estimate with the slit-lamp they were from $5-10\mu$ in diameter, *i.e.*, about the size of, or a little larger than, the nucleus of an epithelial cell.* There was no disturbance of the corneal reflex, the surface being perfectly smooth and bright, so that it seems likely that the particles were intracellular or at least situated in the substance of the corneal epithelium. None was seen deeper than this and most appeared to be in the surface layer of cells.

Across the lower part of each cornea was a series of wavy yellow lines, like a very marked Hudson's line, but wider and brighter than this and branching. This is seen in Fig. 3. The lines were composed of very closely aggregated dots similar to the others over the rest of the cornea, but closer together and much brighter yellow. There was also a more diffuse yellow colour across the cornea in this region, resembling the pale yellow staining of the conjunctiva and suggesting a substance in solution.

The substantia propria was entirely normal, as were the internal parts of the eye. Fig. 4 shows the slit-lamp appearances. The corneal band is on the left and shows the yellow colour of the massed deposits, the general surface speckling and the normal substantia propria. The band of light reflected from the iris shows the yellow dots to be opaque or dark brown on retro-illumination, and to the right of this they are visible as greyish dots seen by scattered light (total internal reflection).

Course and prognosis.—The history of one patient throws some light on the time sequence and on the prognosis. This man,

* The nucleus of a corneal epithelial cell measures roughly $5 \times 7\mu$ and the whole cell $10 \times 18\mu$.

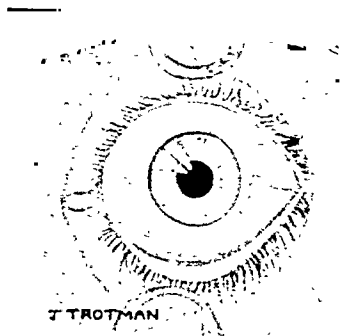


FIG. 1.

Macroscopic appearance of eye of atebirin worker, aged 66 years.

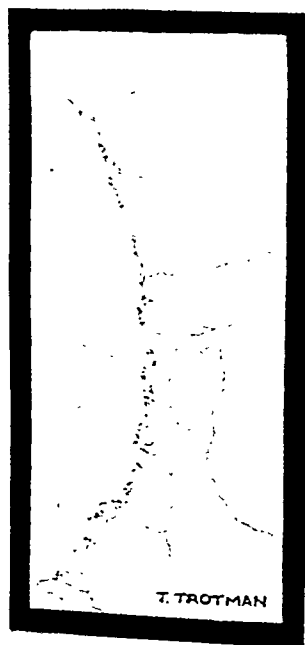


FIG. 2.

Slit-lamp appearance of the limbus of a patient, aged 35 years. (Inset diagram shows areas of distribution of the pigment dots).

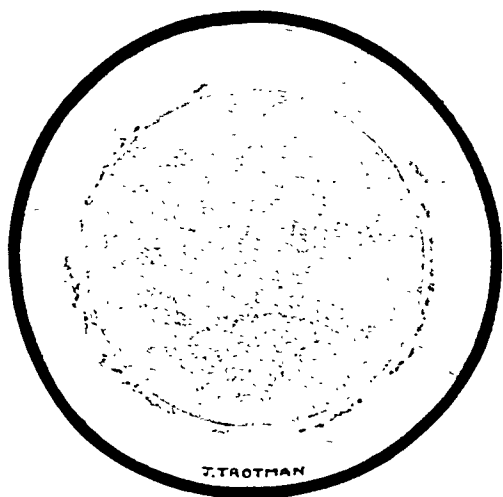


FIG. 3.

Composite slit-lamp drawing of cornea of atebirin worker aged 49 years, showing innumerable yellowish dots and aggregations of these in the lower part. (The dots are not drawn small enough)

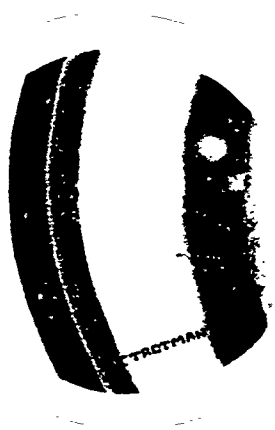


FIG. 4.

Slit-lamp appearance of a portion of the cornea shown in Fig. 1.

J.D., aged 35 years, was first employed on the work two years ago. After a little less than a year he noticed the blue haloes and that the whites of his eyes were yellow. He was then put on light work where he was not exposed to the chemical. After two months of this the "blue rings" disappeared and he considered his eyes quite normal. After a further month, during which he remained well, he went back to work with atebtrin. He remained well for six months and then began to notice faint haloes. These became intensified by nine months, at which time I examined him, but he stated that the rings had not yet reached their previous intensity. He showed the limbal deposits of brown dots and his corneae were very faintly and finely peppered, but there were no yellow lines and the whole appearance was much less marked than in most of the other patients. Without a slit-lamp he would have been passed as normal.

This case seems to show that the condition may be very slow in developing and that if the patient comes off the work it clears away completely, only very slowly coming back on re-exposure. No permanent damage appears to result and the visual acuity is unimpaired throughout. The time for disappearance of the rings after stopping the work is about two months and the time taken to reappear (and to appear originally) is six to nine months.

Differential diagnosis.—The complaint of haloes round lights always arouses a suspicion of glaucoma, but in all these cases the tension was normal, the discs not cupped and the visual acuity 6/6 with full fields. The absence of any signs of glaucoma and the presence of the appearances described above should make the diagnosis simple. In addition, the patients have always been exposed to atebtrin dust for some time before the symptom is noticed.

Discussion of pathology.—It would seem that the punctate deposit on the cornea is the cause of the haloes and that they are produced by a diffraction effect. Three points of interest present themselves for investigation, namely, the route by which the deposit reached the cornea, its nature and its position.

Since the men were exposed to atebtrin dust two routes to the eye were open, either the direct surface involvement, *i.e.* the dust settling on the eye, or the systemic route, the dust having been inhaled, swallowed or absorbed through the skin and passed to the eyes *via* the blood stream or the lymphatic system.

In order to decide this, two observations were made, one on man, the other experimentally on rabbits. In the first, two other men, aged 27 and 36 respectively, were examined. They were volunteers in an experiment on the effects of atebtrin administration in normal people. They had both taken 100 mg. a day by mouth and occasionally more, for seven months and were still taking it when seen. Their

hands and faces and skin generally showed a pale yellow staining, but their conjunctivae were perfectly normal as were also their corneae. They both stated that they had no symptoms, that they had never seen blue haloes, that their sight was unaffected (it was 6/6 right and left in both) and that at no time had the whites of their eyes been yellow. This would seem to indicate that the corneal condition in the atebtrin workers is due to direct surface contamination with very fine dust, which gets embedded in the corneal epithelium and is only slowly cast off.

A second observation to confirm this was made on three rabbits. It was found that atebtrin dust blown directly on to their eyes with an insufflator twice a day produced a similar slit-lamp appearance in six days, with no systemic effects.

It would therefore seem certain that the corneal condition is caused by direct surface contamination with atebtrin.

The particles seen with the slit-lamp cannot, however, be atebtrin itself, since this is completely soluble in the tears. Their exact nature is speculative, but it seems possible that they are precipitates of an insoluble breakdown product of atebtrin formed in the cells of the corneal epithelium which have absorbed the atebtrin itself in solution in the tears. We have some evidence that selective absorption of substances from the tears does take place, but so far as I am aware, no previous instance of intra-cellular precipitation of such an absorbed substance is known.

In order to investigate the exact position of the particles, one of the rabbits mentioned above was killed at the time when the slit-lamp appearance of the cornea resembled that in man. Microscopic examinations of scrapings and flat preparations were made of the corneal epithelium. Yellow granules in the cytoplasm could be seen in unfixed specimens, but fixation with saturated mercuric chloride solution in absolute alcohol and staining with Leishman's stain gave the best results. Granules were then visible, stained faintly blue, and lying well within the cytoplasm of the epithelial cells. Each individual granule was very small and amorphous. Aggregated they formed a granular blue mass either encircling the nucleus or lying beside it, if it happened to be eccentric in the cell. The granules were optically inactive. It is obvious that the opaque yellow dots seen with the slit-lamp are not the individual granules but the whole aggregation within the cell containing them. The nature of the granules is not known. It has been suggested by Dr. H. J. Barber of the Research Laboratories, May & Baker, Ltd., in a personal communication to Dr. Wyers that they may be composed of 2-chloro-7-methoxyacridone, an exceedingly insoluble substance which is slowly formed by hydrolysis of atebtrin. He considers that this substance might well become fixed within cells

and stain them, since both atebrin itself and methylene blue to which it is related have marked staining properties. It is interesting to note that 2-chloro-7-methoxyacridone does not occur in the formula for acriflavine and that no complaints of haloes or of corneal staining have come from acriflavine workers. It does occur, however, in the newer 5-aminoacridine and workers in this material should be watched for development of any signs or symptoms.

That the substance composing the granules is in some way derived from the solution of atebrin in the tears and is not deposited in the cells direct from the dust is indicated by the fact that the granules occur all over the cornea, even under the upper lid. This distinguishes the appearance from that due to a chemical injury with some substance (*e.g.*, mustard gas vapour) which acts directly on each epithelial cell it touches, without going into solution in the tears first.

Once the granules have appeared in the cells they do not redissolve but remain until the cells are cast off and repaired in the ordinary course of events. The affected cells tend to become pushed by the movements of the lids into a more or less horizontal band just below the centre of the pupil (position of Hudson's line) and during the process of recovery (in rabbits) can be detected longest here.

That more severe contamination than that received by the six patients described might produce more severe and permanent results than they displayed was suspected. Two of the rabbits were therefore exposed to large insufflations of atebrin powder twice daily. One of them developed oedema of the substantia propria of the cornea, shedding of the epithelium, marginal vascularisation and iritis. The other showed slight iritis and no corneal oedema. In both the diffuse yellow colour of the atebrin in solution appeared to extend through the whole thickness of the cornea and even to stain the endothelium. Complete recovery took place in both cases on stopping the insufflations.

The nature of the haloes seen by the patients.—The halo is obviously a diffraction effect due to the opaque granules in the epithelial cells. Such haloes are always blue on the inside and red on the outside of the ring. The blue, being brighter, is more readily noticed. Duke-Elder (Text Book of Ophthalmology, Vol. I, p. 801) states "The nature and site of the structures causing the halo can be deduced from the angular diameter of the rings . . . The radius of the halo, divided by the distance of the nodal point of the eye from the light gives the tangent of the angle." An attempt was made to measure the angular diameter of the haloes in this way in three of the men, using a monochromatic filter (transmitting 5500 Å). The results lay between $3^{\circ} 17 \text{ min.}$ and $3^{\circ} 52 \text{ min.}$

Extreme accuracy of measurement was not possible, but the result indicates that the particle size is roughly a little smaller than a normal epithelial cell. The usual angular diameter of the haloes in glaucoma is 7° — 12° and here we know that the size of the droplets causing them is that of a swollen oedematous epithelial cell. Haloes due to mucus on the corneal surface are larger still (up to 14°).

An attempt was also made to calculate the actual particle size by measuring the haloes seen with monochromatic filters (5500\AA and 5400\AA) and using the formula

$$d = 1.22 \times \frac{\lambda l}{r}$$

where d = diameter of particle

r = radius of first dark ring in cm.

l = distance of eye from screen

λ = wave-length in μ

the actual measurements were difficult to do accurately and the results of a number of trials gave values for d varying between 7μ and 11μ . This, though not conclusive, points to an intra-cellular cause for the diffraction effect and would seem to correspond with the massed amorphous particles.

The investigation of this new industrial disease is chiefly of interest from the physico-chemical point of view and from the importance of the differential diagnosis of glaucoma which its history suggests.

Summary

A new industrial disease of the cornea is described in atebtrin workers. It appears to be caused by an intra-cellular deposit of an insoluble derivative of atebtrin.

Its only symptom is the seeing of coloured haloes (mostly blue) round lights. Its prognosis is excellent on removal from contact with atebtrin dust.

SUBJECTIVE "LIGHTNING STREAKS"*

BY

R. FOSTER MOORE

LONDON

IN the October, 1935 number of this Journal I published a series of cases of a symptom complex under the above title (p. 545), and later, an additional series in *The American Journal of Ophthalmology*

* Received for publication, November 21, 1946.

(November, 1940, Vol. XXIII, p. 1255). Professor F. H. Verhoeff has published an excellent account of them in *The American Journal of Ophthalmology* (March, 1942, Vol. XXV, p. 265).

My reason for reverting to the subject is, that I am now able to add the personal experiences of the symptoms in three ophthalmic surgeons, which may, on this account, be thought to have particular value; they compel me to modify, and enable me to amplify my former description in several prominent particulars.

Professor Verhoeff has been good enough to send me a detailed and lucid account of his experiences, and to make suggestions which, coming from him, have unusual authority; Dr. J. Rutter Williamson sends me an excellent description of his observations, and these have a value of their own from the fact that he sees with one eye only; and since my last paper I have myself become a subject of the "Streaks." I propose to describe the condition in the light of the above additional evidence.

This is a symptom complex which occurs after middle age—the youngest of my patients was 42—most usually in myopes, and perhaps more commonly in women.

It consists in the association of the development of flashes of light with the simultaneous appearance of spots before the eyes. I previously expressed surprise that patients should be sufficiently concerned to seek advice on account of these symptoms alone, as they sometimes will, but having experienced them myself, I can understand that they should arouse a degree of curiosity, if not of actual anxiety as to their significance, for the phenomenon is quite a striking one.

The most conspicuous feature is the sudden appearance, without obvious cause, of bright or brilliant flashes of light.

Various similes have been made use of in describing them, but as "lightning" is the most usual I adopted it.

Amongst other similes are the following:

J. R. W. says "On the evening of November 25, 1942, I noticed flashes after switching off the light to go to bed. I thought there must be a defect in the "black-out" showing searchlights, but investigation proved negative."

F. H. V. says "I have found on close questioning that patients all agree that what they see are streaks."

For myself, were I experiencing the phenomenon as a quite unfamiliar sight, I feel no doubt that I should have compared the streaks to lightning.

Others have suggested "shooting stars"; "fireworks"; "headlights," etc.

Few have commented upon the colour, but J. R. W. describes a "bright bluish yellow" flash: one person spoke of "silver lightning,"

and one compared the flashes to fire-flies, but said they were too silvery in colour.

They are of momentary duration so that detailed analysis of them is not possible; they are very bright and travel from above down. They are curved as, I fancy, lightning never is; they do not exhibit the zig-zag feature of lightning.

J. R. W. says "It commenced in the upper part and ran downwards, always in a perfectly true semi-circle"; this too is true of what I see. They may be seen at any time, though naturally they are more conspicuous in the dark; they occur whether the eyes are open or closed.

Position of the streaks.—My original statement as to the part of the field in which the streaks are seen, was that they were "almost always to the outer side of the eyes" and whilst this is an accurate statement they certainly do occur at other positions.

F. H. V. says:—"The streaks in my left eye began about October 30, 1937. For a short time they occurred in all quadrants. In 1939 I could elicit them only in the outer quadrant. On October 19, 1942, I first observed the streaks in the right eye; at first here too they were in all quadrants and I could sometimes get an almost complete circle of light; all but the lateral streaks, however, ceased to occur in about a month. At the present time (May 24, 1946), I can still elicit a temporal streak in either eye."

J. R. W. says:—"It was situated to the outer part of the field (he having only one seeing eye, the left). On its disappearance there swept in from the right, *i.e.* from the nasal side, a sort of secondary flash, a wavy cloud of pearly colour, very like what is seen with the ophthalmoscope sometimes, in a detached retina. This wavy appearance was trembling, as if a very thin cloud disturbed by a current of air; the whole was of course of extremely brief duration. Very rarely did one see a straight flash, and then it was very small and appeared on the nasal side of the field of vision.

My own experience is that the streaks are seen only on the rather extreme temporal side, and do not transgress a central vertical limiting line, but at times I have seen what I can only describe as faintly luminous circular areas to the nasal side.

It can be said that the streaks may be seen in any quadrant of the field, but that they are much more often referred to the temporal side, and that here they persist much longer than elsewhere.

Movement of the eyes. I am surprised that in my former account I had not elicited that the flashes are only seen on movement of the head or eyes, a fact which is pointed out by Prof. Verhoeff. Had I at that time been the subject of them, I could not have overlooked the fact; most folk are agreed that they can produce them at will by sudden movement of the eyes.

Verhoeff says:—"I can elicit both streaks by quick rotation of

my head. This of course causes an ocular rotation to the opposite side, so that the resultant is simply a 'sideways motion of the eye as a whole.'

J. R. W. says—"I could produce it at will by moving the eye quickly to the left."

For myself I can say that while the eyes are at rest the flashes are never seen, and that I can elicit them, not with certainty but quite frequently, by sudden movement of the eyes.

It is unnecessary to labour the fact that movement is necessary for their production, the only point upon which I have some doubt is as to whether movement of the head will do so; I think it likely that it will, though I have not been able to satisfy myself as to this.

Verhoeff's explanation (referred to later) of the method of production of the streaks clearly calls for movement, whether of the head or eyes or both.

It is quite clear that the streaks do not occur with the eyes at rest.

Unilaterality. I find that in three cases only of my first twenty-six was the condition bilateral. In Verhoeff's case streaks appeared in the second eye about five years after the first; he says, with true prescience, "probably you will later get streaks in your other eye," this occurred just a year later.

I think it likely that most cases become bilateral in time.

Persistence. My original statement that the streaks persisted for "varying periods up to two or three months" needs revision.

Of the present three observers F. H. V. tells me (May 24, 1946), "The streaks have persisted about nine years in my left eye, and about four in my right; I still can produce them at will in both eyes, but only in the outer field of each."

J. R. W. says:—"They only lasted in their original brilliance for six weeks or so; after two months I occasionally see the semi-circular flashes but not brilliantly illuminated as before."

For myself, they first appeared in the right eye on October 17, 1943, and in the left eye on October 20, 1944; I still see them in the temporal field of each eye, *i.e.*, after three years in the right and two in the left eye; they are less obtrusive than formerly and I think occur less frequently, though no doubt one has got used to them and notices them less.

In view of the above it is clear that one can put no limit to the period for which they may occur, and it seems likely that, in some cases at least, they may persist indefinitely, especially in the temporal fields.

The association with vitreous opacities. Of the close relationship between the occurrence of these streaks and the simultaneous development of vitreous opacities I have no doubt.

It may well be that the patient has been familiar with opacities for many years, and in this case the development of streaks is

heralded by the sudden appearance of a fresh crop of them, or it may be that the two, the streaks and the opacities make their first appearance at the same time, *i.e.*, within a few hours of each other; in any case the association is very constant and I do not think these characteristic streaks occur without the development of a crop of opacities.

F. H. V. says:—"My vitreous opacities are much greater than they were before the streaks first appeared."

J. R. W. says "In daylight one was constantly seeing showers of black spots in a smoky environment and these moved across the page when reading."

For myself, as a low myope, I have been familiar with muscae for many years. On the evening of October 17, 1943, whilst out walking, I noticed a new, rather conspicuous unfamiliar spot, in the lower temporal field of the right eye, it was oat-shaped and of a rather golden colour; twenty four hours later I saw, for the first time, bright flashes, running from above down, curved, vertical in direction and well to the temporal side.

On October 20, 1944, flashes, having the same character, suddenly appeared to the temporal side of the left eye, and in the evening, a shower of dark opacities appeared.

The immediate cause of the streaks. I believe Verhoeff's explanation of the immediate cause of the streaks is probably the correct one; he attributes them to a shrinking and partial separation of the vitreous which then impinges upon the retina on movement, he says—

"To explain the more frequent occurrence of the streaks in the outer field, it is necessary to assume that the separated vitreous is more apt to strike the nasal retina, or that the sentient part of the retina extends further forward on the nasal side, or that both of these conditions obtain. Since the vitreous is asymmetrical, in the sense that the disc is nasal to the posterior pole of the eye, the separated vitreous would be nearer to the retina on the nasal side, and it is, of course, a known fact that the visual field extends furthest on the temporal side."

At a later date he writes:—"I can elicit both streaks by a quick rotation of my head—this fact confirms my view that a streak is due to the impingement of the separated vitreous upon the retina. You should try again to elicit the streak in your eye by a quick rotation of your head."

Up to date, I have come across no case which causes me to modify my belief that the streaks have no sinister meaning, either at the time, or, what is more important, ultimately.

A SURVEY OF THE RESULTS OF LACRIMAL
STRICTUROTOMY *

BY

IVOR LLOYD

BRADFORD

ALTHOUGH the writer knows of no series of published cases illustrating the point, it is well recognised that the treatment by probing of epiphora due to disease of the lacrimal duct is extremely unsatisfactory. Ainsler has even said it may be easier to cure a detachment than a simple epiphora.

Stricturetomy for the relief of obstruction at the neck of the lacrimal sac is an established technique in French clinics. It is associated with the names of Poulard, Hartmann and Bastan.

English surgeons have not been interested to any great extent in the procedure and there do not appear to be publications of the results of a composite series of stricturetomy operations.

The writer, therefore, decided to give the method a trial on a reasonable number of cases, both from the point of view of verifying the results of the French ophthalmologists and because it presented interesting possibilities.

Method

The French method, used in the Hôpital Lariboisière, and explained with great clarity by Bastan, consists of opening the lower canaliculus with a Weber, Dugast, or similar knife, for 3 mm., and then dilating the canaliculus with a number 3 sound; a knife with a bent handle is used where there is undue projection of the eyebrows. The stricturetomy knife was then passed down into the sac and engaged in the neck of the duct. It was then verticalized, after which the edge was rotated forwards through an angle of 45° so that it faced somewhat forwards to prevent cutting the lower part of the canaliculus at its opening into the sac. Gum-elastic sounds from number 11 to 14 were then passed into the duct and the largest one left *in situ* for 10 minutes. Dilatation was continued at intervals of four to six weeks; the largest viable sound again being allowed to remain *in situ* for 10 minutes to stretch the opening.

The writer found that, with practice, the canaliculus could be dilated sufficiently to allow the passage of the knife with a slight or negligible slit. This was, in fact, preferable, as there was a considerable risk of occlusion of the punctum and/or the canaliculus from scarring if it was cut with a knife.

Dilatation was performed every two weeks for two months and then discontinued unless indicated by a return of symptoms.

Analysis of the Cases

There were 31 patients which involved 33 stricturotomies. In order to give the method an extensive and searching trial a wide variety of types were included, the only exceptions being very old subjects or those in whom there was an excessive amount of purulent regurgitation. The ages of the patients were between 20 and 55 years. The duration of the symptoms averaged 2 years, although one or two patients had had epiphora for 10 years or more. One case was the result of extensive burns of the face which produced scarring of the lids and caruncle, but the canaliculi remained patent.

Mucoceles were present in four cases, and twelve of the patients had chronic low grade muco-purulent infection of the lacrimal passages. Ten of the patients had bilateral obstruction, and in two of them the bilateral operation was performed.

In all cases the condition of the nasal passages and sinuses was investigated.

Lipiodol X-ray photographs of the lacrimal passages were taken in some cases before and after the operation in order to determine the effects of the stricturotomy. It is regretted that this could not be done in all of the cases owing to the pressure of work and shortage of films in the war period.

Results

The French have claimed 100 per cent. cures in cases with no bony obstruction and 70 per cent. in those with bony obstruction. When pus or induration was present the results were correspondingly 3 to 7 per cent. less favourable.

Four of the writer's cases could not be traced later, so that the effective total of stricturotomies for analysis was 29.

Complete cure resulted in 15 of the patients (52 per cent.); "improvement" was obtained in 7, *i.e.*, patients considered the result of the treatment worth while. The percentage of symptomatic relief satisfactory to the patient was therefore 76 per cent.

There were seven failures. An analysis of these showed the following details:—

1. Impassable stricture, probably bony. This is unfortunately not diagnosable pre-operatively.

2. & 3. Very tight strictures in which the stricturotome was passed with difficulty, dilatation being impossible later. Complete cure was obtained in both by the dacryocystorhinostomy operation of Toti.

4. One case was successful from a surgical point of view, and the sac and duct syringed through quite easily five months after the operation, but despite the absence of any other abnormality of the lacrimal passages the epiphora recurred three months later.

5. In one subject infection was a feature with moderate mucopurulent regurgitation. The operation of stricturotomy and subsequent dilatations kept the epiphora and infection in check, but they recurred at the termination of the treatment.

6. The patient with the scars due to burns was shown to have a medium sized mucocele on radiological investigation, but stricturotomy was unsuccessful as a lacrimal abscess developed shortly after the treatment had commenced, and the method had to be abandoned.

7. As a matter of interest and to produce visual evidence of the presence of a stricture, the writer performed an open stricturotomy on one subject. Continuous dilatation was maintained for a week after the operation by a bougie, by which it was hoped to avoid periodic dilatation later. Although the minimum of trauma was inflicted and the sac was closed with very fine cat-gut sutures through the superficial layers only, the subsequent scarring at the lower end of the canaliculus and in the sac led to a return of the epiphora some months afterwards.

Discussion

The operation of stricturotomy has many advantages over the usual methods of treatment of lacrimal obstruction.

It can be performed in the out-patient clinic, thus avoiding the necessity for admission to the wards of a hospital. It is a relatively minor operation leaving no residual unsightly scarring of the face. If the method fails it does not prejudice the exhibition of any other surgical procedure on the lacrimal passages. Finally, it is of especial value in cases where the obstruction is of a fibrous type in which ordinary probing so often proves unsuccessful.

The writer, in agreement with the French clinicians, maintains that the results are as good, if not better, than those obtained with other methods.

On the other hand, the operator must be prepared to devote at least one half day a week to the "lacrimal clinic" if many cases are to be treated, and the large number of dilatations involved renders the method rather tedious. In addition, despite the use of local anaesthesia, dilatation was a slightly painful procedure. It was considered inadvisable to inject an anaesthetic on each occasion owing to the soreness of the skin produced.

Considering the above objections the writer was pleasantly surprised to find that the patients were anxious to continue the treatment, indicating their appreciation of the relief of symptoms.

The presence of a stricture at the junction of the lacrimal sac and the naso-lacrimal duct has been denied, but the writer has shown by radiological findings and also at the open operation that such an abnormality exists in many cases of epiphora. After passing a

stricturotome it was quite evident that some constriction had been relieved because a large probe or bougie, previously impassable, then entered the duct without undue force being required to make it do so.

Summary

The results of 33 stricturotomy operations are analysed, and the causes of failure detailed.

Modifications of the French technique are suggested.

Its advantages compared with other methods of treating lacrimal obstruction are discussed.

The writer wishes to express his grateful appreciation to Mr. John Foster of Leeds, for introduction to the method and the reference quoted, and correspondence on the subject, also for permission to publish these cases treated in his clinic.

REFERENCE

BASTAN, N.—*Ann. d'Ocul.*, 171, Part 1, January, 1934.

ANNOTATIONS

Areopagitical

"Areopagitica: a speech for the liberty of unlicensed printing" was published by Milton in 1664 as an answer to an order in Parliament that "no book, pamphlet, or paper should be henceforth printed, unless the same was first approved and licensed by such, or at least by one of such, as shall be thereto appointed.

"Give me," said Milton, "the liberty to know, to utter, and to argue freely according to conscience, above all other liberties.

This was the beginning of a campaign for freedom of the press which ended in 1695, when William the Third ratified the bill for ending censorship. Everyone who writes or publishes owes a debt to Milton. Had we been editing the British Journal of Ophthalmology three hundred years ago we tremble to think of what might have happened to us after the publication of any particular number. Doubtless we should have been acquainted with the pillory, our nose might have been slit, our ears cut off, we might have been flogged at the cart tail from our domicile or consulting room to the printers' offices every other month; and the fines demanded of us would have soon landed us in the Fleet, or debtor's prison. But, happily for us, these barbarities are a thing of the past. And though

the censorship was revived during the War years, these penalties were not. This Journal was indebted to the Censor and his department for advice and assistance on many occasions.

Milton's sonorous sentence might well form the motto of all printing offices.

Mister or Doctor

In England it has long been the custom to address a surgeon as "Mister." This doubtless had its origin in times when surgery was practised only by the barber surgeons and was untouched by physicians. Samuel Johnson derives the word "mister" from the French word for a craft, *mestier*, the mystery of —, in our case the mystery of the barber surgeons. In 1215 Pope Innocent III prohibited ecclesiastics from undertaking surgical operations on the view that "Ecclesia abhorret a sanguine." However, the church in later years took to burnings and all the horrors of the Inquisition.

Towards the end of the last century some difficulty in this matter of address arose with the advent of the specialties which require both surgical and medical attention, for instance, gynaecology. At first a gynaecologist was called "Doctor," more recently the younger generation seem to prefer "Mister." In Scotland, and indeed elsewhere in the world, ophthalmologists are addressed as "Doctor," and this seems more proper than "Mister," for in the case of most ophthalmologists the surgical work of their practice is appreciably less than the medical work they do.

The pathologist retains the title of "Doctor" and this differentiates his academic and medically qualified status from the laboratory technician who in these democratic days may insist upon the address of "Mister" instead of "George." With the approaching state service and its plans to include optical practitioners (it seems that this is the last title they prefer to "consulting ophthalmic optometrists," "ophthalmic opticians," and so on) that in England we might conform to the custom of other countries and call the medically qualified eye practitioner "Doctor." Strictly speaking medical men should not be called "Doctor" unless they possess this University degree (which many ophthalmologists do) but it has been the practice in this country to accord this as a courtesy mode of address to any qualified man or woman practising medicine, regardless of whether the qualification is a bachelor of medicine and surgery or a diploma of the Colleges of Physicians and Surgeons. It would seem that now is the time to decide this point of whether the professional address of ophthalmologists might be altered to "Doctor."

THE CONTACT LENS SOCIETY

A NEW Society, purely scientific in object and organisation, has been formed for the study of contact lens work in all its aspects. Members may be medical or non-medical, and it is hoped to stimulate interest in the subject among optical workers, doctors, physiologists and others whose domain touches the question at any point.

The Secretaries of the Society, Mr. A. G. Cross and Mr. G. H. Giles, will be glad to supply information and forms of application for membership on receipt of a request addressed to 65, Brook Street, London, W.1.

The first scientific meeting will be held on January 27, 1947, at 5.30 p.m., at the headquarters of the British Optical Association, 65, Brook Street, London, W.1. Thereafter it is hoped to hold scientific meetings four times a year, or more often if sufficient material is forthcoming.

As will be seen, membership of the Society requires nomination by three members of the Council. For the benefit of those wishing to join, officers of the Society are as follows:—

President: Professor Ida Mann; *Vice-Presidents:* Mr. F. A. Williamson-Noble and Mr. K. Clifford Hall; *Joint-Secretaries:* Mr. A. G. Cross and Mr. G. H. Giles; *Treasurer:* Mr. C. H. Keeler.

Other members of the Council are:—Messrs. J. H. Doggart, G. B. Ebbage, F. Juler, Sir Stewart Duke-Elder, Messrs. F. Dickinson, H. B. Marton, G. D. McKellen and T. Hamblin.

The Rules of the Society are as follows:—

1. *Object of the Society.* The object of the Society is the scientific study of contact lens work in all its aspects and the promotion of research into its various problems.

2. *Meetings.* Meetings shall be held as and when the Council deem necessary.

3. *Constitution.* Membership shall be open to anyone holding a medical or scientific qualification and engaged on work connected with the subject, provided that he is satisfactory to the Council and is willing to declare himself bound by ethical rules of a scientific Society. These are:—

- (a) To keep no process or formula secret in any communication.
- (b) To allow other members of Society to see records and to repeat experiments.
- (c) Not to use membership of the Society as an advertisement.
- (d) Not to use the Society as a forum for advertisement of commercial processes or preparations.
- (e) To agree to answer to the best of his ability all scientific questions put by fellow members of the Society.

Such persons shall be ordinary members of the Society. The Council shall be nominated from among the members and shall contain equal numbers of medical and non-medical persons, the latter to include at least one representative of the dispensing opticians. There shall be 14 Founder Members nominated by the Contact Lens Sub-Committee of the Faculty of Ophthalmologists. They shall retire at the end of two years and subsequently annually, and shall be eligible for re-election at the Annual General Meeting.

4. *Officers of the Society.* The officers of the Society shall consist of a President, two Vice-Presidents, two Hon. Secretaries, an Hon. Treasurer and not less than eight other members of the Council.

5. *Election of Officers.* The officers of the Society shall be elected yearly by ballot, after the first two years, at the Annual General Meeting. A balloting list of the names recommended by the Council, from their number, for election shall be posted to each member two weeks previously.

6. *Admission of Members.* Subsequent members shall be proposed by three or more members, but can only be admitted by a unanimous vote of the Council. Applicant should state the nature of his connection with and interest in contact lens work, and his intention to abide by the ethical rules of the Society.

7. *Subscriptions.* The annual subscription shall be initially £1 1s. 0d. per year. Any member whose subscription is twelve months in arrears shall, at the discretion of the Council, cease to be a member of the Society.

8. *Duties of Officers.* The President shall regulate the proceedings of the Society and Council and enforce observance of the rules.

One of the *Vice-Presidents* shall deputise for the President in his absence.

The *Hon. Secretaries* shall conduct correspondence, keep minutes of meetings, notify new members of their election, notify the Hon. Treasurer of resignations or death or changes of address. They shall arrange the meetings with the President, and shall arrange for the publication of papers communicated, subject to agreement with an Editorial Committee.

The *Hon. Treasurer* shall receive money, make payments and keep accounts. The accounts shall be audited and presented at the Annual General Meeting. The Auditors shall be nominated at the Annual General Meeting.

9. *Meetings of Council.* Council shall meet as and when deemed necessary and not less than three times per year. Five members shall constitute a quorum. The President shall have a casting vote in addition to his ordinary vote. The Council shall decide upon all questions relating to publications and communications and shall have

power to submit any paper to referees. A summary of all intended communications must be submitted to the Council before being read.

10. *Vacancies of Officers.* Vacancies on the Council may be filled by co-option during a current year and by nomination and election at the Annual General Meeting.

11. *Publication of Papers.* After being passed by the Council, publications may be submitted to the appropriate journals by the author and Hon. Secretaries in consultation.

12. *Notice of Meetings.* One month's notice of meetings shall be sent to each member.

13. *Visitors.* No representatives of the press shall be admitted. Each member may bring one visitor to one meeting only.

14. *Funds.* It shall be in the power of the Council to allocate grants from the funds of the Society for research, but such grants must be approved by a General Meeting of the Society.

BOOK NOTICES

Physiologie Oculaire Clinique. By A. MAGITOT. Editors: Masson et Cie, Paris. Pp. 458, with 235 figures. 1946.

Ophthalmologists in every country in the world will be interested to know of a book on the physiology of the eye written by Magitot of Paris, for in our generation he is one of the few masters in this branch of our subject who has spent a great part of his life exploring its many illusive and fascinating problems. For a quarter of a century he has been a systematic explorer in this wide field and whenever questions arise regarding the intra-ocular pressure, the ocular circulation or the reflexes governing the activities of the eyes, the opinions of Magitot must always be consulted and are usually respected. It is good that he has had the opportunity to set out and analyse our knowledge as it stands to-day in a comprehensive and authoritative volume which will disappoint few who read and study its pages.

In its scope the book is comprehensive and takes in all aspects of the physiology of the visual system—the eye, its central connections and the higher perceptual centres. The first chapter deals with the protective mechanism—the lids and lacrimal apparatus; the second with the sensory (trigeminal) mechanism. The following three chapters deal with the nutrition of the eyes, the vascular circulation, the nature of the aqueous and vitreous, and the factors controlling the intra-ocular tension. In view of Magitot's classical contributions to this subject it is not surprising that these are among the fullest and best chapters in the book. The theory of dialysation of the

intra-ocular fluids is accepted in its totality and a strong case made out for its validity. Then follow chapters on the physiology of the conjunctiva, the cornea, the iris and pupillary reactions, the lens and the retina. The physiology and pathology of the pupillary reactions are unusually well described, and the account of the physiology of the retina and of vision is of more than usual interest. Magitot adopts the somewhat original treatment of describing the dual activities of the retina quite separately—the “diurnal” retina comprising the function of the cones and colour vision, and the “nocturnal” retina dealing with the rods, the visual purple and dark adaptation. There follows a most interesting discussion of the optic pathways. The more recent work of Adrian, Hartline, and particularly of Granit is well summarized and a strong case put forward for the presence in the optic nerve and visual paths of fibres with different functions, not only visual and pupillary fibres, but among the former a further differentiation of fibres carrying impressions of light, form and different colours. The chapter on the higher organisation of vision at the cortical level deals with the recent knowledge contributed by the neuro-surgeon to the functions of the brain and discusses the various types of hemianopias, cortical blindness, psychical blindness, the visual agnosias and disorders of disorientation; the author in this part of his book follows closely the teaching of Lhermitte. The final section dealing with ocular movements and their central nervous organisation and with binocular vision is the least authoritative and original in a volume which covers a range of subjects so vast that the standard cannot be reasonably expected to be on the same level. Physiological optics are entirely omitted.

The book is written not by a pure physiologist but by a clinician, and one of its best features is the stress laid upon clinical questions and the intimate relation of physiological principles to clinical problems. The applications of physiology to pathology are constantly stressed, and this approach to clinical pathology constitutes one of the most useful aspects of the book. Some of the most recent works on the vegetative physiology of the eye and on the neuro-physiology of vision are not mentioned, but presumably current literature was difficult to obtain in France during the war. Bibliographical references are limited to the more important contributions and are by no means comprehensive.

On the whole this is a good, if not an excellent volume which will find a welcome and respected place in ophthalmic literature. The author is to be congratulated on his industry, his philosophy and the clarity of his exposition—to read the book has been a pleasure. And the publishers are to be congratulated on the maintenance of their high pre-war standard in the paper, printing and illustrations of what is quite a luxurious volume.

Transactions of the American Ophthalmological Society. Vol. XLIII. 1945.

The Eighty-first Annual Meeting of the American Ophthalmological Society was held at The Homestead, Hot Springs, Va., on November 12, 13 and 14, 1945. Vol. XLIII of the Society's Transactions contains 591 pages devoted to original communications. The first of these, by Colonel Derrick T. Vail is a charming narrative of his experiences as ophthalmic consultant to the American Forces in England and later in France.

We hope to publish abstracts of the other papers in the abstract section of the British Journal of Ophthalmology. This volume also contains lists of the Society's Officers, Council, past Presidents, members and the recipients of the Howe Medal.

This volume is of the usual high standard that characterizes these Transactions. Its production in printing, paper and illustrations is good.

Transactions of the Ophthalmological Society of the United Kingdom, Vol. LXV. London: J. and A. Churchill, Ltd. 1946.

The 65th volume of the *Transactions* is a much more bulky article than its immediate predecessor. It contains the list of communications read at the Annual Congress in May, 1945, together with the report of the joint clinical meeting of the society with the Section of Ophthalmology of the Royal Society of Medicine, the proceedings of the Oxford Ophthalmological Congress and those of the Irish Ophthalmological Society. Many of the papers are of considerable interest and of permanent value; such as the reports of the discussion on the ocular sequelae of head injuries, the Doyne Lecture and A. J. Mooney's paper on the visual manifestations of head injuries. A colour plate illustrates O. Gayer Morgan's paper on a case of crush injury to the chest associated with ocular complications. We hope that by the time Vol. LXVI is published the rest of the affiliated societies will have resumed their work; and that the volume as a whole will have got back to pre-war size. At the same time a word of congratulation is due to the Society, and particularly to Mr. Frank Law, for the way in which the sequence of 65 volumes has been unbroken in spite of the difficulties of the past seven years.

Instrumental Optics. By G. A. BOUTRY. Paris: Masson et Cie., 1946.

There is no such thing, says Boutry, as a *complete* treatise on instrumental optics. However far the technique of construction may advance, some margin of instrumental error will persist, and even if a perfect optical engine could be designed, the manifestations

of its activity would still have to filter through the interpretative mist of the human eye. Indeed the voice of scientific authority is gentler than it was two generations ago. The grim, rationalistic monuments of science erected in the nineteenth century have crumbled away, to be succeeded by the rarified world of quanta and electrons, more subtle and fantastic than webs spun by philosophers.

The most enchanting trait of present-day physicists is their readiness to waive old and treasured hypotheses. We are apt to take for granted the new doctrine that light does not travel *only* in straight lines, and consternation is no longer registered at the failure of thermodynamic laws to account for all the phenomena. It was not always so. Nor are old theories the only ones now to be abandoned. What happens when the enlightened modern scientist has to face facts that conflict with Planck's principle of the discontinuity of radiant energy? He gaily falls back upon electromagnetic undulations as a means of restoring continuity, and he denies the possibility of synthesising the truth about luminous energy in one comprehensive statement. What a contrast with Victorian science, which not only reconstructed monsters from single bones, but even achieved the regimentation of atoms!

If any one is tempted to boast of his facility with the microscope or with any other up-to-date optical device, he should surely call to mind the accomplishments of our forefathers, who pursued such detailed observation in spite of their crude appliances. Boutry recalls how Leeuwenhoek succeeded in discovering spermatozoa by means of a seventeenth-century loupe. Since then a vast number of refinements have been evolved. The modern lens is elaborately contrived to avoid marginal astigmatism, field curvature and other sources of distortion, but the necessary calculations are rendered comparatively simple by experience handed down through several centuries. The author of *Instrumental Optics* fully acknowledges his debt to the pioneers, as well as to his contemporary colleagues.

Prisms, cylinders and the optical faults of the human eye are considered at some length, but Boutry does not profess to describe the minutiae of ocular anatomy and physiology. The fifth and last part of the book consists of a systematic, well-illustrated survey of instruments, including telescopes and spectroscopes. Considerable attention is also focused upon the optical problems of microphotography, but the most detailed study of all is reserved for the microscope. The author is to be congratulated on a skilfully planned book, which represents a notable addition to the long list of scientific achievements already standing to France's credit.

NOTES

Honours

THE Société Belge d'Ophtalmologie has made Sir Stewart Duke-Elder an honorary member.

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Western Ophthalmic Hospital, Annual Report, 1945

THE Ninetieth Annual Report of the Western Ophthalmic Hospital shows that since the cessation of hostilities the out-patient attendances and admissions to the wards have both increased. It is hoped to adapt the former quarters of the nursing staff for the use of in-patients. Extra accommodation is urgently needed in view of the long waiting list. Most of the honorary staff, who were on war service, are now returned. Details of finance are appended.

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Ophthalmological Society of the United Kingdom. Annual Congress, 1947

THE Annual Congress of the Ophthalmological Society will be held in Glasgow, on March 27, 28 and 29, 1947.

The President's address is entitled: "De Senectute."

The subject for discussion will be "Rhinology in relation to Ophthalmology" which will be opened by Dr. John Marshall (Ophthalmic Surgeon), Mr. Gilbert H. Howells (Ear, Nose and Throat Surgeon), and Dr. R. McWhirter (Radiologist). Members who desire to take part in the subsequent discussion are not required to intimate their intention before the Congress. No member may speak for more than ten minutes.

Members wishing to read papers are asked to send the titles to Mr. E. F. King, 79, Harley Street, W.1, as soon as possible. Abstracts of papers, which will be circulated at the Congress and subsequently to the leading ophthalmological journals abroad, should be submitted not later than January 31, 1947.

On Friday afternoon there will be a clinical meeting at the Glasgow Eye Infirmary.

On Saturday afternoon visits have been arranged to the Corporation of Glasgow Art Galleries, Kelvingrove, under the guidance of the Director Dr. T. J. Honeyman, and to the Hunterian Museum of the University of Glasgow, by invitation of the University Court.

Members who wish may stay at the University Hostel for men, Maclay Hall, 17, Park Terrace, Glasgow, C.3. Accommodation consists of rooms for two or three. The inclusive cost of bed and breakfast for the three days will be one guinea. Towels cannot be provided. Dr. J. D. Fraser, Tennent Memorial Institute of Ophthalmology, Western Infirmary, Glasgow, W.1, is prepared to make reservations at Maclay Hall, or at the Glasgow hotels. Members are invited to write directly to Dr. Fraser as soon as possible as hotel accommodation is severely limited.

The Annual Dinner of the Society will be held on Thursday, March 27.

Post-graduate
Course in
Ophthalmology

THE George Washington University Department of Ophthalmology is planning the resumption of the Wm. Thornwall Davis Intensive

Post-graduate Course in Ophthalmology, February 3-8, 1947. These courses have been given annually except during the war when they were temporarily discontinued.

The following guest lecturers will participate:—Dr. F. Heed Adler, Philadelphia; Dr. J. Moson Baird, Atlanta, Ga.; Dr. S. Judd Beach, Portland, Maine; Dr. Hermann M. Burian, Boston; Dr. Ramon Castroviejo, New York City; Dr. C. Alvin Clapp, Baltimore, Md.; Dr. F. Bruce Fralick, Ann Arbor, Michigan; Dr. Dean B. Judd, Washington, D.C.; Dr. Peter C. Kronfeld, Chicago; Dr. Walter I. Lillie, Philadelphia; Dr. Angus L. MacLean, Baltimore, Md.; Mr. Philip L. Salvatori, New York City; Dr. Harold C. Scheie, Philadelphia; Dr. Edmund B. Spaeth, Philadelphia; Dr. Frederick W. Stocker, Durham, North Carolina. Subjects of clinical interest to the practising physician doing eye work will be presented.

The 10th Annual Post-graduate Course in Ocular Surgery, Pathology and Orthoptics will be given during the week of January 27-February 1, 1947. The Army Institute of Pathology will give the instruction in pathology under the direction of Colonel J. E. Ash, Medical Corps, U.S.A., Scientific Director of American Registry of Pathology National Research Council. Colonel Ash will be assisted by Helenor Campbell Wilder, Ophthalmic Pathologist, Army Institute of Pathology; Lawrence P. Ambrogi, Chief of Laboratories, Army Institute of Pathology; Dr. M. Noel Stow, Dr. Arnold W. Forest and Miss Eleanor Paul.

The Surgery demonstrations will be given by the resident staff under the direction of Dr. Ernest Sheppard, Professor of Ophthalmology. This is a practical course with demonstrations on animal eyes.

For further details of the Post-graduate Courses in Ophthalmology write to the Secretary, Suite 34, 1801 K Street, N.W., Washington 6, D.C.

* * * *

Ophthalmological
Society of Egypt

THE Annual Meeting of the Ophthalmological Society of Egypt will take place at the Memorial Ophthalmic Laboratory, Giza, on Friday and Saturday, March 14 and 15, 1947, at 9 a.m. Medical practitioners, oculists or otherwise, are cordially invited.

* * * *

Copyright material

A MEMORANDUM regarding the use of copyright material has been sent to us by the Publishers' Association Medical Group.

It is pointed out that in recent years, with the development of photographic methods of reproduction, the use of copyright material without permission has greatly increased.

Infringement of copyright is discussed under the following headings: "Digest" journals. "Abstract" journals. Films. Microfilms. Film strips, which are also known as film slides. Lantern slides. Mimiographic reproduction. Illustration, and lastly, photo-stats.

The legal position is as follows: "The reproduction without the consent of the owners of the copyright of any protected work or any substantial part thereof in any material form, is an infringement of copyright. The only exceptions, material for the present purpose are two in number.

- (a) "If a teacher were to use an epidiascope to throw on the screen in front of his class an illustration from a copyright book there would be no infringement; but if he were to make a film slide of the illustration for use in the same way he would be infringing the copyright."
- (b) "Photographs of sculpture or craftsmanship permanently situate in a public place or building do not involve an infringement of copyright."

There appears to be very little piracy of complete books in this country; but it seems that a certain amount of unauthorised reproduction of such parts as parts of the text, tables, illustrations and bibliographies has increased of late years.

The matter concerns both author and publisher and it is suggested that:

- (a) Copyright material be not used without permission.
- (b) Where such material is used, due acknowledgment is made to the source from which it is taken and "arrangements agreed as to the manner in which copyright material is used."
- (c) Reasonable payment is made where the copyright material is to be put to a commercial use and in certain circumstances, *e.g.*, where the proposed use of the copyright material would be damaging in some way to the Publisher's and/or Author's interest.

Neither Publisher nor Author is any way inclined to take a dog-in-the-manger attitude, but the question needs to be regularised.

* * * *

Corrigendum IN Mr. Spencer Walker's paper on Myopia and Pseudomyopia, December, 1946, p. 739, para 2, line 5, for "temporarily" read "eventually."

THE BRITISH JOURNAL OF OPHTHALMOLOGY

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FEBRUARY, 1947.

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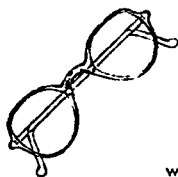
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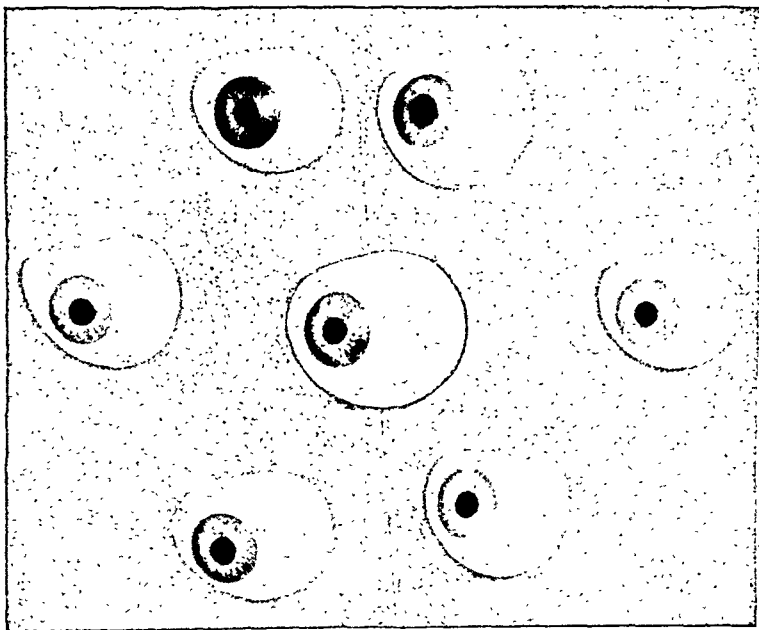
THE BRITISH JOURNAL OF OPHTHALMOLOGY LTD.

24-27, THAYER STREET, W.1



ARTIFICIAL EYES IN PLASTIC

Artificial Eyes until the beginning of the war were invariably made of glass, and their production was entirely in the hands of a few very skilled craftsmen in this Country and on the Continent. Up to August, 1939, Theodore Hamblin, Ltd., employed Mr. Paul Asprien, of Vienna, at 15, Wigmore Street, and at their various provincial branches, where he made artificial eyes in glass while the patient waited. With the outbreak of war, these visits had to cease and steps were taken to develop the manufacture of artificial eyes in plastic material. The many difficulties of producing eyes in this material have been overcome, and they are now made throughout in plastic, no paper or glass being incorporated.



Eyes made in plastic have many advantages over those made in glass. They are life-like in appearance, comfortable in wear, are not affected by the secretions of the orbit, and above all, they are unbreakable.

Difficult shapes necessitated by war injuries, burns, etc., or thin shells to fit over shrunken or deformed globes, almost impossible to produce in glass, are quite possible in plastic.

Patients may be sent in to 15, Wigmore Street or to most of the provincial branches, where stocks of ready-made eyes are available from which selections may be made and fitted. Specially made eyes for more difficult orbits can be made with little delay. For these a carefully made mould of the orbit is first taken, and a special iris is produced in plastic. In such cases a second visit after the eye has been made is necessary for fitting.

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THE BRITISH JOURNAL OF OPHTHALMOLOGY

FEBRUARY, 1947

COMMUNICATIONS

THE PRODUCTION OF A FILTERING CICATRIX IN GLAUCOMA*

BY

SIR RICHARD CRUISE

LONDON

At long last I have obtained for microscopic examination an eye on which I had performed a successful hinged flap sclerotomy 12 years previously. Mr. Eugene Wolff has cut sections which substantiate the theory on which I have operated for the last 25 years.

The theory was that it was possible to establish permanent drainage from the anterior chamber by means of incisions alone, without excision of any tissue.

This drainage was arrived at by enlisting the assistance of the endothelial cells in the formation of a filtering cicatrix (Fig. 6):

1. The technique consists in dissecting a conjunctival flap up to the limbus as in trephining.
2. A triangular flap of corneo-scleral tissue is cut with its blunt apex attached at the limbus.
3. The conjunctival flap is replaced but *not* sutured.
4. Primary union of the corneo-scleral flap is prevented by massaging aqueous through the incision under the conjunctiva.

* Received for publication, December 14, 1946.

which causes the corneo-scleral flap to ride up and delay approximation of its edges.

5. This massage must be done before 24 hours have elapsed, and continued for a week or ten days, otherwise there will be a tendency to premature union.

6. Primary union is delayed deliberately to enable endothelial cells to proliferate and insinuate themselves between the cut margins at the expense of the normal C.T. cells and form a permeable track through the periphery of the anterior chamber (Fig. 6). It follows then that the greater the length of incision through endothelial lined tissue, compatible with safety from iris prolapse, the more consistent will be the result. My experience is that incisions amounting in linear measurement to 9 mm., formed by a blunt apexed triangle with a 5 mm. base and lateral cuts 2 mm. give the best results.

In the *Trans. Ophthalm. Soc. U.K.*, 1940, I described the operation I performed for many years, using a keratome and angled knife for the lateral cuts (Figs. 2 and 3). There was frequently some difficulty in making the lateral cuts owing to the mobility of the small amount of tissue involved and other manipulations, and, in fact, a colleague admitted to me that though appreciative of the results obtained, he did not adopt my method because it was *too difficult*.

As difficulty in the performance of an operation is a serious handicap to its popularity, I have evolved the following technique, which demands the minimum of digital dexterity or risk (Figs. 4 and 5):

1. A flap of conjunctival and subconjunctival tissue is dissected down to the limbus, splitting the cornea for $\frac{1}{2}$ mm.

2. A special hook with a very sharp point is inserted into the corneo-sclera just above the limbus, so as to get a firm hold of the eyeball.

3. The anterior chamber is entered by dissecting through the sclera $1\frac{1}{2}$ mm. from the limbus with a scalpel or Graefe knife for a distance of 5 mm. If the aqueous is permitted to escape slowly the iris does not prolapse. If it does, it can be replaced with a repositor and there will be no tendency for iris protusion when the posterior chamber is empty.

4. While the corneo-scleral lip of the incision is held by the hook, with a pair of sharp-pointed scissors two lateral cuts of 2 mm. are made, one at each end of the incision, and sloped towards each other, so as to make a blunt angled hinge, with the apex towards the cornea.

5. The conjunctiva is replaced but not sutured. If the conjunctival flap is thick, as it is in some cases where there is plenty of subconjunctival tissue, a suture that merely keeps the flap from flopping forward may be inserted, but never pulled tight, as this

would tend to hasten healing. By this means an incision of 9 mm. has been made in endothelial lined tissue, and my experience is that this is adequate to produce an endothelial paved cicatrix with minimal risk of prolapse of the iris.

The late Colonel Herbert in 1907 introduced his operation of flap sclerotomy, whereby he obtained in a certain percentage of cases a filtering scar and permanent reduction of tension. Unfortunately, there was a larger percentage in which no filtration took place, and on account of the unreliability of the results, Colonel Herbert gave it up, and in 1921 substituted a form of iris inclusion with a cystoid scar.

Colonel Herbert explained the results he had obtained in flap sclerotomy as due to a shrinking of the tissue between his incisions, and persevered with what he described as "wedge isolation" for the purpose of obtaining consistent shrinking. The wedge, he stated, "may be likened to a disconnected graft of fibrous tissue . . . and like any completely separated graft, may be confidently expected to shrink in bulk. The shrinkage should provide for filtration." Herbert's operation with its incisions, which were varied with much ingenuity, was unsuccessful because the basic principle, "shrinkage of tissue," was incorrect.

FIG. 1.

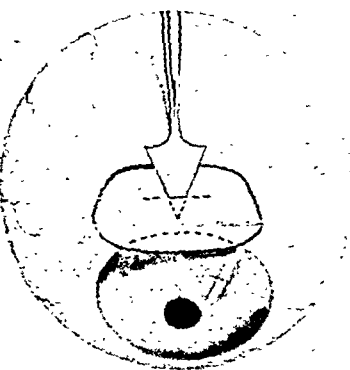


FIG. 2.

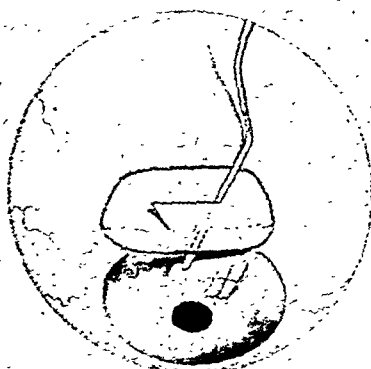
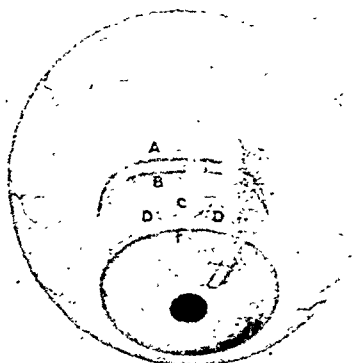


FIG. 3.



The question of endothelial cells being responsible for the filtration was never considered, and as far as I am aware endothelial activity has never before been taken into account and deliberately utilised.

Some years later, after an interlude of enthusiastic trephining for glaucoma, my satisfaction with that operation was shaken by two cases of late infection in my private practice. I therefore tried Herbert's operation, without arriving at any consistency in results, while aiming at shrinkage of the isolated wedge of tissue. However, in one case on which I had performed a flap sclerotomy, and which had firmly healed at the first dressing, I opened up the incision subconjunctivally with a repositor, and repeated the performance on two succeeding days. I found that the oedematous area over the incision remained boggy, and was undoubtedly permitting leakage of aqueous. It was obvious that prevention of union and delay in healing was the key to the situation. The only explanation that seemed feasible was, that there must be outgrowth of endothelial cells that would allow aqueous to percolate through the incisions and form a filtering cicatrix (Fig. 6). Later on, in the *Trans. Ophthal. Soc. U.K.*, 1940, before I obtained microscopical proof, I submitted that it was a race between the endothelial and connective tissue cells, and the type of scar, filtering or impermeable, depended on which type of cell predominated in its formation.

I was impressed by a paper by Thomson Henderson, in the "Ophthalmoscope," 1907, on "So-called filtering cicatrices in the treatment of glaucoma." He poured scorn on the possibility of such an occurrence, stating that "the endothelium seals the inner margin of the incision and that it is this endothelium that precludes all possibility of a permanent filtering cicatrix following any operative incision, however devised." This statement was histologically correct for normal healing.

However, accompanying his paper is a very instructive illustration of the normal healing of a limbal incision taken from a case that died suddenly 8 days after operation. This shows the endothelium growing forward into the irregular posterior margin of the corneal incision to meet the fibrinous plug that was forming primary union. It occurred to me that if this fibrinous plug could be prevented from consolidating and interference with normal healing deliberately achieved, enterprising endothelial cells, unable to bridge a gap, would tend to grow into, and line, the gap. My aim has been to prevent normal healing by deliberately keeping the incisions apart; this is achieved by massage, which expresses the aqueous under the conjunctival flap, and thereby raises the hinge flap of corneo-scleral tissue with its endothelial lining. This gives time for the endothelial cells to proliferate and line the margins of

the incisions, and instead of "sealing" off the incisions, renders them permanently permeable.

Now that there has been histological verification of my clinical hypothesis, I feel I am justified in strongly advocating the hinge flap sclerotomy as the operation of choice in cases of chronic glaucoma.

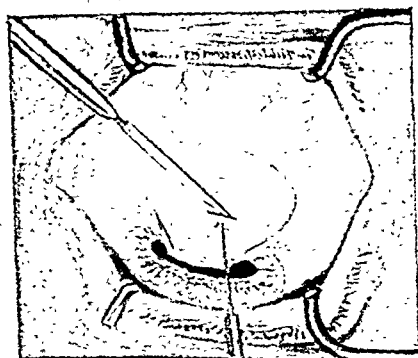


FIG. 4.

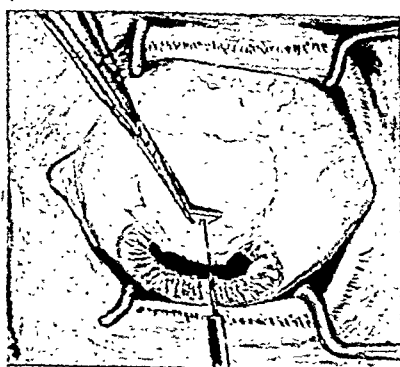


FIG 5.



FIG 6.

Scheme as suggested to R.C. to indicate method by which sclerotomy produces permanent drainage. Note endothelial lined track.

For 25 years I have aimed at utilising the endothelium of the anterior chamber as my accomplice in committing an offence against nature, that is, the prevention of the normal healing by primary union of every incision into the anterior chamber, without excision of any tissue.

The evidence supplied by the regular periodic visits of cases in one's private practice where alone accurate tonometric observation



FIG. 7.

Low power view of the area of drainage, with a portion of the track leading from it.



FIG. 8.

Portion of Fig 7 under higher power.

can be maintained over periods varying from 6 months to 25 years, fortifies me in my conviction that the "offence" has been justified.

I have to thank my late House Surgeon, Mr. Dermot Pierse, for his excellent drawings of the operation, and for his comment, "I hope your paper will have the desired effect of changing the views of the many die-hard trephine enthusiasts. I am very satisfied with the results I obtain from the operation."

Mr. Boxill, another House Surgeon, is not less enthusiastic. Evidence from successive generations of House Surgeons, with

their varied experience of various surgeons, and their ill-concealed intolerance of any misadventures by their seniors, is not a negligible criterion.

The eye which is the subject of histological investigation by Mr. Eugene Wolff, was obtained by a most fortuitous chain of circumstances within 3 hours of the death of the patient. It was noted at the time of the removal of the eye that aqueous could be expressed from the anterior chamber into the subconjunctival tissues. The eye was fixed in Zenker and serial sections cut in celloidin. The sections were stained with haematoxylin and eosin, van Gieson, Mallory's triple stain, Mallory's phosphotungstic acid haematoxylin and Verhoeff's elastic tissue stain.

The boggy area of filtration is covered by thinned conjunctival epithelium containing two to four layers of nuclei (Figs. 7 and 8). It is limited above by a characteristic papillary-like thickening of the epithelium. The drainage area itself is largely filled with a very delicate spongework of connective tissue with here and there some fine fibres which stained like Descemet's membrane.

The actual track in the corneo-sclera also was not empty as might be thought from haematoxylin and eosin stained sections. But sections stained with Mallory's triple stain show it to be filled

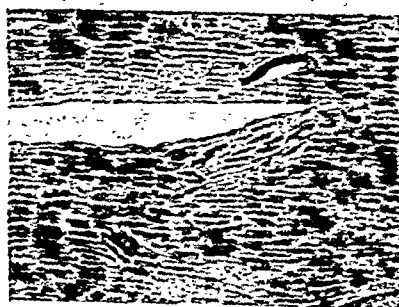


FIG. 9.

Drawing of a portion of the track to show endothelial lined and bare areas.

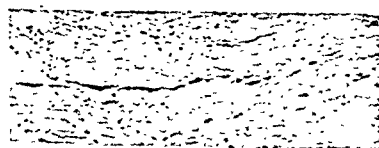


FIG. 10.

Microphotograph of portion of the track to show endothelial lining.

with the same spongework of very delicate connective tissue as the drainage area. Here and there also are some spots of pigment.

The walls of the track are in part lined by very definite endothelium (Figs. 9 and 10), while in others the sclera is bare, and it is a very remarkable feature that where the sclera is bare it has apparently undergone no change since the day the incision was made some twelve years ago.

In many of the sections a portion of Descemet's membrane is seen (Fig. 9). This is no doubt due to the fact that Descemet's membrane tends to curl forwards when cut.

Comment

An endothelial lining to filtering scars is usually denied but there can be no doubt of its presence here. The fact that the sclera has shown no signs of healing appears to bear out Thomson Henderson's theory that tissues bathed by the aqueous are not stimulated to show the usual signs of repair.

THE FLAP SCLEROTOMY IN THE TREATMENT OF GLAUCOMA*

BY

W. M. DE C. BOXILL

LATE R.M.O., ROYAL WESTMINSTER
OPHTHALMIC HOSPITAL

SINCE the introduction of a similar operation by the late Colonel Herbert in 1907, but based on an erroneous hypothesis, the technique of the flap-sclerotomy appears to have been considerably varied by those few who, in the past, have used it in the treatment of chronic primary glaucoma. The technique, which will later be described, has arisen through the work of Sir Richard Cruise at the Royal Westminster Ophthalmic Hospital during his long and successful association with that hospital, and it is through the efforts of this great master of the simple extraction operation for cataract that an even simpler method of permanent decompression of the hypertensive eye has been evolved.

In the past this operation has mostly been confined to cases of chronic glaucoma particularly the primary ones. Recently it has been performed with success on acute congestive hypertensive eyes which did not respond to medical treatment. The technique in both

* Received for publication, January 2, 1947.

cases being the same with the pre-operative treatment varied accordingly.

The factors in favour of this operation apply equally to both acute and chronic cases which require operative treatment. They are as follows:—

(a) This is the least traumatic of all the operations for ocular decompression. No tissue is excised. An iridectomy is unnecessary and therefore avoided, hence there is no haemorrhage into the anterior chamber with the subsequent passage of blood admixed with aqueous into the sclero-corneal wound thereby facilitating its blockage and early closure:

(b) Slow decompression of the anterior chamber:

(c) The permanent incisions into the angle of the anterior chamber are covered by a flap of conjunctiva and Tenon's capsule and therefore protected from the conjunctival sac. The advantage of this in the acute case with a potentially dirty conjunctival sac is clear;

(d) It is an easy operation to perform.

The pre-operative treatment is slightly different in the two types of case. In the primary acute eye, operative treatment is generally adopted when the pupil and tension do not respond to intensive treatment with miotics, heat, etc. It seems likely that here the corneal oedema is so great that miotics are unable to pass through the oedematous barrier in sufficient concentration to be effective. At this stage, when it has been decided to adopt surgical measures, a retro-bulbar injection of 1 c.c. of 3 per cent. novocaine is given through the lower lid with a $1\frac{1}{4}$ " needle and the commencement of the operation delayed until the tension is lowered sufficiently for the corneal oedema to clear and the pupil to respond to miotics which are now able to pass through the cornea in effective concentration, also the conjunctiva by now is less congested and the slight bleeding which is subsequently encountered at operation is easily controlled. Furthermore, as the cessation of pain rapidly follows the retro-bulbar injection, the apprehension of the patient is lessened and the operation can proceed under conditions approximating those prevailing with a cold case.

If 1 c.c. of 3 per cent., novocaine is not sufficient, which is rare, the injection can be repeated giving $\frac{1}{2}$ c.c. at a time until the condition of the eye is such that operative procedure can be undertaken under ideal conditions.

As the conjunctival sac is potentially dirty, just prior to the operation it is washed out with a sterile warm solution of normal saline and guttae penicillin 500-1,000 units per c.c. in normal saline instilled. Penicillin drops can also be used during the period of medical treatment with miotics, etc.

With regard to the giving of a retro-bulbar injection to an eye

whose extra-ocular tissue is very congested, there are some who would say that the dangers of intra-orbital haemorrhage following this injection are greatly increased when it is given in this type of case, however, assurance has been obtained by no less an authority than Mr. Eugene Wolff of the Royal Westminster Ophthalmic Hospital, that there is no increase in the occurrence of intra-orbital haemorrhage providing the injection is given with a needle not more than $1\frac{1}{2}$ in. in length and in practice, so far, this dreaded complication has not arisen.

In the case of chronic primary glaucoma, the pre-operative requirements are both few and simple. Eserine $\frac{1}{2}$ -1 per cent. is frequently instilled into the conjunctival sac 1-1 $\frac{1}{2}$ hours before the operation so that the pupil is as small as possible and well under the influence of this drug, also, guttae pantocaine 1 per cent. at frequent intervals $\frac{1}{2}$ - $\frac{3}{4}$ hour before the operation to ensure good anaesthesia.

In the theatre $\frac{1}{2}$ - $\frac{3}{4}$ c.c. of 3 per cent. novocaine is injected sub-conjunctivally in and around the tendon of insertion of the superior rectus muscle. The advantages of this injection are:

(a) Paralysis of the superior rectus muscle to prevent the patient looking upwards during the operation.

(b) Increase of the anaesthesia of the conjunctiva and Tenon's capsule in the immediate area of operation.

This injection is not usually required when dealing with an acute case if the retro-bulbar injection has been given into the muscle cone around the ciliary ganglion and time enough has been allowed to elapse for its anaesthetic and paralytic action to have full effect. However, $\frac{1}{4}$ - $\frac{1}{2}$ c.c. of 3 per cent. novocaine with 1 minim of adrenalin 1/1,000 added may be injected sub-conjunctivally around the insertion of the superior rectus muscle in those acute cases where the conjunctival vessels are excessively congested, in order to reduce bleeding when the conjunctival flap is being cut.

The technique of the operation is as follows:

A small snip is made into the conjunctiva and Tenon's capsule about 6 mm. directly above 12 o'clock on the cornea, this snip is then extended medially and laterally and for an equal distance on both sides of the original snip so that an incision is obtained which is roughly 1-1 $\frac{1}{2}$ cms. in length and is parallel with the limbus. This flap composed of the above two structures is then turned downwards and gradually dissected off the sclera using non-toothed forceps for fixation and blunt nosed conjunctival scissors for the dissection until about 2 mm. from the sclero-corneal junction, when sharp-pointed scissors are substituted to complete the dissection. The actual snips made in this region should be as near to the sclera as possible; if not the conjunctiva may be button-holed, for in this region the conjunctiva, Tenon's capsule and episcleral tissue are

fused and therefore not as easily raised from the sclera as has been previously encountered. During this part of the dissection the sclero-corneal junction is reached and recognised by the sharp demarcation of the grey tissue of the cornea and of the white sclera; the junction where these two meet may be referred to as the "grey line."

When the "grey line" is reached, which usually first appears in the centre of the dissection, the dissection is then carried laterally and medially with sharp-pointed scissors so that the "grey line" is exposed for about 4-5 mm. As soon as this step has been completed, a Tooke's corneal splitter is used to carry the dissection forwards in the same plane in order to separate the corneal tissues the whole length of the exposed "grey line" and for a distance of $1\frac{1}{2}$ mm. in front of it; the raised portion of corneal tissue remaining in continuity with the previously raised conjunctival flap above and the cornea below.

The above dissections having been completed, oozing episcleral and conjunctival vessels should be dealt with in order to provide a clean dry field in preparation for the incisions now to be made into the scleral and corneal tissues. The conjunctival flap is now turned downwards on to the cornea with its conjunctival surface in apposition with the anterior surface of the cornea: the assistant steadying the flap with a small swab soaked in normal saline and held between the tips of non-toothed forceps; the swab being applied to the Tenon surface of the flap, traction being gently applied in a downward direction, but without exerting pressure on the eye.

There is now an area of the outer surface of the angle of the anterior chamber exposed for the permanent incisions which are to be made in this situation, but before commencing these incisions, it is essential that perfect fixation of the eye is assured. Of the many methods of fixation, the aim of which is to render the eye immobile without exerting external pressure upon it, the one which has been found to answer these requirements is by the use of the scleral hook, whereby the superficial scleral fibres are picked up by passing a small sharp pointed hook beneath them. The hook most commonly used in this operation is the Cruise scleral hook (John Weiss and Co.) which has been specially designed for this operation.

The next step in the operation is the insertion of the scleral hook, the place of insertion being along the "grey line," the points of entrance and exit of the hook being approximately on either side of, and equidistant from, 12 o'clock on the cornea, the distance between these points being about $1-1\frac{1}{2}$ mm. With the scleral hook *in situ* and held by the free hand of the operator, an incision is now made with a small scalpel or Graefe knife into the sclera 5 mm. long and 1 mm. from and parallel to the "grey line," the mid-point of the incision being approximately opposite 12 o'clock on the cornea.

Since the sclera extends for a distance of 2 mm. from the anterior extremity of the ciliary body to the sclero-corneal junction in this upper portion of the anterior chamber, the base incision just mentioned is therefore placed 1 mm. in front of the root of the iris, so that neither this tissue nor the ciliary body is in danger of injury when the knife enters the anterior chamber; furthermore prolapse of the iris is less likely to occur than if the incision was placed further back. As the aim of the incision is to make a linear opening into the angle of the anterior chamber the whole thickness of the sclera has to be traversed throughout the entire 5 mm., hence it would be impossible to complete it with one stroke of the knife without sudden entry into the anterior chamber with consequent injury to the iris and particularly the lens, therefore, the incision is made with a series of cuts all in the same place and all of the same length, until the anterior chamber is entered, this usually taking place by a small opening in the centre of the wound. The cutting edge of the knife should *always be perpendicular* to the antero-posterior curve of the sclera to avoid making a sloping incision with bevelled edges which tends to separate the superficial scleral fibres of the anterior portion of the wound which would make for difficulty in completing the inner portion of the incision.

The anterior chamber now having been entered at a small point in the centre of the wound, there is a slow leakage of aqueous from it and therefore slow decompression is achieved. As soon as the anterior chamber has been decompressed, the point of the knife is inserted into the small opening with the cutting edge directed upwards and the inner portion of the incision completed medially and laterally.

It is during the decompression of the anterior chamber and the subsequent enlargement of the inner portion of the incision that the complication of prolapse of the iris may occur; but providing the knife is sharp and little pressure has been used in making the cuts, the use of the scleral hook for fixation to avoid further pressure on the globe and the sphincter pupillae is well under the influence of the previously administered miotic, this complication seldom arises. If it does occur the iris can usually be reposed and this should be done before continuing. Very occasionally the iris will not remain inside the anterior chamber after many repositings; in this event a peripheral iridectomy will prevent the iris from making further appearances into the lips of the wound; however, as this complication will not arise if the above precautions are taken, one of the previously mentioned main points in favour of this operation will not have been missed.

Fixation still being maintained with the scleral hook *in situ*, but now held by the assistant, the operation is continued by making two forward cuts in the sclero-corneal tissue; these are made with small

sharp pointed straight or curved scissors. Each of these forward cuts commences at the extremities of the original incision making an angle with it of approximately 45 degrees and extending forwards for 2 mm., that is, 1 mm. of sclera and 1 mm. of cornea compose each cut. A triangle comprised of the two tissues is thus formed with a 4 mm. base of scleral tissue and two sides each 2 mm. in length containing equal portions of sclera and cornea. The distal ends of these two forward cuts do not completely meet so that the apex of the triangle is not completed and a small portion of corneal tissue is left in continuity with the rest of the cornea, resulting in mobility of the triangle only in a vertical direction.

Just prior to commencing these forward cuts the tip of an iris repositor is kept between the lips of the base incision by the free hand of the operator until the cuts are completed in order:

(a) to separate the lips of the incision so that the point of the scissors may be introduced into the wound without difficulty when commencing and completing each forward cut,

(b) to keep the iris away from the point of the scissors,

(c) to ensure against the iris prolapsing at this stage.

In those cases where there are peripheral anterior synechiae at the apex of the triangle these adhesions may sometimes be broken down with a repositor; at other times the adhesions may require division with scissors, thereby freeing the iris from the posterior surface of the cornea. A repositor should then be passed into the anterior chamber to ensure that free communication has been established between it and the triangular opening in its angle. The operation is completed by removing the scleral hook and repositing the conjunctival flap under the upper lid. No sutures are inserted into this tissue so that no tension is exerted upon it with subsequent pressure and closure of the triangular valve beneath it.

The immediate instillation of eserine $\frac{1}{2}$ per cent. in both acute and chronic cases prior to bandaging the eyes, is in the opinion of the writer a safeguard against prolapse of the iris during the 24 hours before the first dressing when the anterior chamber is attempting to reform. Atropine sulphate 1 per cent. should be instilled at the first dressing and at successive 24 hourly dressings until digital massage of the globe is commenced; the use of atropine here being solely to raise the intra-ocular pressure behind the mobile triangular flap of sclero-corneal tissue, thereby helping to keep it open.

The time to institute gentle digital massage being when the conjunctival flap has completely sealed off and there is no leakage of aqueous into the conjunctival sac; this may be ascertained by the daily instillation of fluorescein. If massage is employed before, there may be a sudden rush of aqueous through the permanent

incisions taking with it the iris and probably necessitating further surgical interference in the form of an iridectomy. On the other hand once the conjunctival flap has healed, there is sufficient back pressure on the aqueous in the anterior chamber to prevent its sudden escape through the triangular opening, there is now no danger of the iris prolapsing.

As soon as massage has been started it may be carried out once or twice daily during the post-operative period to ensure the passage of aqueous through the opening and to prevent healing occurring at its edges. The patient is allowed up at this juncture and can usually be discharged around the tenth day. He is instructed to massage the eyeball gently daily through the lower lid and is seen at weekly intervals until it has become obvious that a permanent filtering cicatrix has been produced; at this stage massage is discontinued.

OPHTHALMO-NEUROLOGICAL SYMPTOMS IN CONNECTION WITH MALIGNANT NASOPHARYNGEAL TUMOURS*†

BY

DR. ERIK GODTFREDSSEN

COPENHAGEN

From Radiumhemmet, Stockholm (Professor L. Berven), Kung Gustaf V'tes Jubilaumsklinik, Lund (Professor L. Edling), and the Radiological Clinics of Copenhagen (Physician-in-Chief, Jans Nielsen) and Aarhus (Professor C. Krebs).

MR. CHAIRMAN, Ladies and Gentlemen,

I feel greatly pleased and honoured to be allowed to speak before this dignified audience and thus to re-establish the connection between British and Danish ophthalmology, a connection which was previously so intimate and profitable. Danish ophthalmologists, among whom the best known are Bjerrum, Tscherning, Edmund Jensen, Heerfordt, Lundsgaard, and Roenne, are greatly indebted to British ophthalmologists, whose best known representatives with us are men like Young, Mackenzie, Bowman, Priestley-Smith, Marcus Gunn and Duke-Élder.

During the gloomy years of German occupation Danish ophthalmology greatly missed the inspiration from British ophthalmology. Now that I stand here as the first Danish speaker after the war I

* Read before the Ophthalmic Section, Royal Society of Medicine, London, November 14, 1946. † Received for publication, December 6, 1946.

naturally call to mind the heroic contribution made by the English towards the liberation of my native country; for which we are all very grateful, and this will never be forgotten.

Realising the importance for us in Denmark of the re-establishment of this connection I thank you for your kind invitation to the present meeting, and I hope I shall not betray the trust reposed upon me by the Society.

As the subject of which I want to speak has been dealt with but rarely in British literature, I hope to be able to bring forward for discussion facts which are little known to you.

Introduction

An account of the ophthalmo-neurological symptoms in connection with malignant nasopharyngeal tumours must necessarily include a description of other important features of the symptomatology to give the right idea of the position in the nosography of the ophthalmo-neurological symptoms.

An increased interest, though varying in degree, has been taken in malignant nasopharyngeal tumours since the introduction, about 100 years ago, of the postrhinoscopic speculum examination, through which it has become possible to diagnose diseases in the nasopharynx. This interest has been further stimulated within the past few decades on account of the results obtained from modern radiological treatment, which to-day is capable of curing these deep-seated and generally inoperable tumour forms. The increased interest is, however, due to other facts as well, thus for instance to the fact that these tumour forms have proved to be of more frequent occurrence than they were previously supposed to be, and that the disease is very often misjudged.

The cause of this misjudgment may to a certain extent be explained from the variety of the clinical picture, which but rarely conveys the idea of a malignant neoplasm in the nasopharynx. That a no small number of patients are young persons often in sound general health likewise contributes to this fact.

The widely different symptoms or combinations of symptoms imply, besides applications to general practitioners, also examinations of the patients by various specialists often at rather early stages, thus by ophthalmologists, neurologists, oto-rhinologists, surgeons, neurosurgeons, internists and radiologists.

If the idea of a malignant nasopharyngeal tumour in the case in hand does not occur to the examining physician and the time for the making of the right diagnosis is delayed, by which valuable—perhaps life-saving—time is wasted for the patient.

Historical data

I shall abstain from a detailed historical review of the literature in hand. Only I want to mention that the nosographical pioneer works are due chiefly to French writers (Escaï, Laval, Jacod), and in England to Trotter, while most of the modern comprehensive descriptions come from America (New and others).

Nasopharyngeal tumours were the order of the day at the International Medical Congress in London, 1913. But very little was said about ophthalmological symptoms.

Frequency, histopathology, age and sex incidence

The malignant nasopharyngeal tumours, though of somewhat infrequent occurrence are by no means rare. Thus over 2,000 cases have now been published in the literature, and personally I managed to collect 454 cases over a period of ten years from the four Scandinavian radiological clinics. The following numerical statements are based on these latter cases.

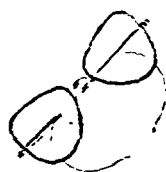
The four radiological clinics from which the cases under review are derived are as follows: *Radiumhemmet*, Stockholm and the Radiological Clinics of Lund, Copenhagen; and Aarhus, which together cover a population corresponding in size to that of London (about $7\frac{1}{2}$ million people). The frequency of malignant nasopharyngeal tumours constituted here 1 per cent. of the total number of cancer cases, *i.e.*, a frequency of the same relative magnitude as that of choroidal melanosarcoma. As, however, only the cancer cases that are likely to respond to irradiation are referred to the radiological clinics, we must make a correction for this percentage to attain to the frequency among the total number of cancer cases in the population. The figure then arrived at is 0.4 per cent. of all cases of cancer.

A bare two-fifths (38 per cent.) of the patients presented ophthalmo-neurological symptoms, a figure which varied but little from one year to the other. —

Histopathological examinations revealed both carcinomata and sarcomata, in conformity with the fact that malignant nasopharyngeal tumours proceed from the mucous membrane and the lymphatic tissue in the nasopharynx. I shall not go into details regarding these facts, but only just mention that the majority of the tumours were anaplastic tumours of low differentiation. Most common were squamous epithelial carcinomata and reticulum-cell sarcomata, which occurred equally often. The undifferentiated tumours and those of low differentiation constituted four-fifths of the cases, while the differentiated tumours, *i.e.*, squamous epithelial carcinomata undergoing cornification constituted one-fifth.

As to the *age incidence* (Fig. 1) all age-classes from 4 to 79 were represented, but half of the patients were found within the age-classes of 41 to 60. It is, however, worth noting that *one-fourth of the patients were 40 years of age or younger*. The average age was 49. When cases of carcinoma and sarcoma were taken separately the average age was somewhat higher for carcinomata than for sarcomata.

For no discoverable reason there were twice as many men as women both among the carcinoma cases and among the sarcoma cases.



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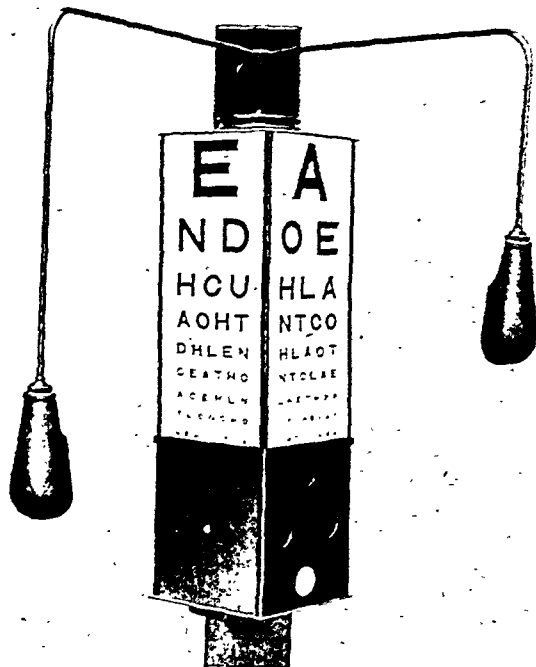
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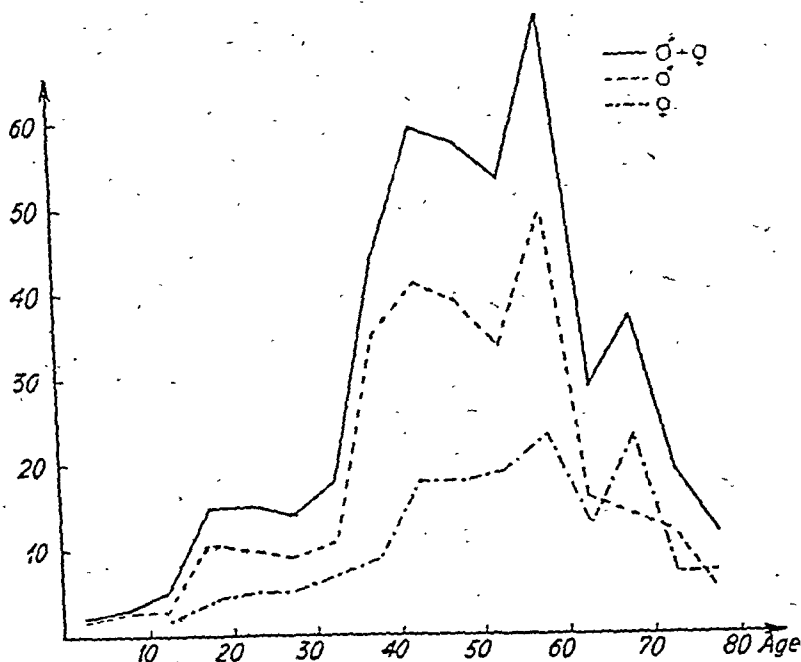


FIG. 1.

Age-curves for 454 cases of malignant nasopharyngeal tumours.

Symptomatology

The symptomatology can roughly be divided into four well-characterised groups:

- (1) *Ophthalmoneurological symptoms* due to the infiltrative growth of the primary tumour or its metastases into the base of the skull and adjacent regions,
- (2) *rhinological* and
- (3) *otological symptoms* due to the exophytic growth of the primary tumour into the nasopharynx with influence on the Eustachian tube,
- (4) *enlarged cervical glands* in consequence of lymphogenous metastases.

I shall here give a brief survey of the manner of growth of the tumours and of the topographical anatomy of the affected areas, because the understanding of the symptomatology of the malignant nasopharyngeal tumours, in particular the ophthalmoneurological symptomatology, rests to a great extent on a knowledge of these facts.

The most frequent *point of origin of the primary tumour* is the lateral wall of the nasopharynx, especially the lymphatic tissue of the tubal tonsil (half of the cases). Next in frequency follows the

roof (the nasopharyngeal tonsil, one-third of the cases), and finally the posterior wall (Fig. 2).

A characteristic feature of the malignant nasopharyngeal tumours is their tendency to *infiltrative and destructive penetration*

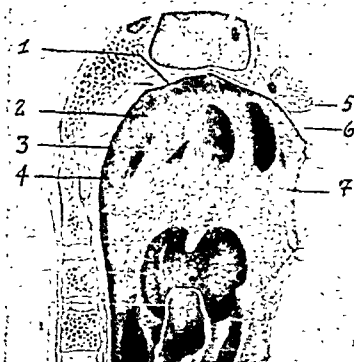


FIG. 2.

The nasopharynx seen from behind and from the right side with the characteristic relief: pharyngeal tonsil (1), tubal tonsil (2), tubal ostium (3), Rosenmüller's pharyngeal recess (4). The soft palate (7) and the posterior border of the nasal septum (5) bound the choanae (6). (After Testut).

into the adjacent areas, partly the *parapharyngeal space* and partly the *base of the skull*. The tumour either follows the foramina already present or grows with osseous perforation. The parts of the base that are particularly exposed to invasion are (Fig. 3)

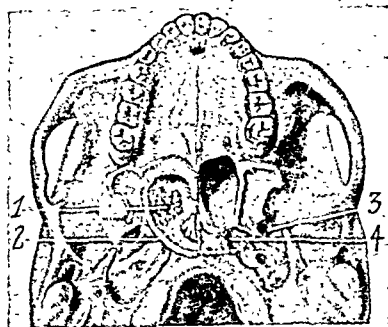


FIG. 3.

The limit of the nasopharynx on the external base of the skull appears from the dotted white line running between the foramen lacerum (4) and the foramen ovale (3). To the left in the picture the nasopharyngeal wall is drawn (2) with a neoplasm arising from the tubal ostium (1). (The Fig. modified after Canuyl).

naturally those which lie along the line of demarcation between nasopharynx and base of the skull, which is marked by the place of insertion of the pharyngo-basilar fascia. This area is seen to

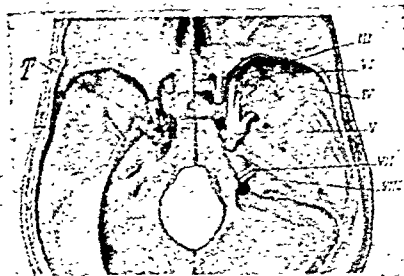


FIG. 4.

Internal base of the skull. The Roman numerals indicate the numbers of the brain nerves. To the left (T) there is seen a nasopharyngeal tumour invading the middle cranial fossa through the foramen lacerum (*Canuyl*).

comprise parts of the body of the sphenoid bone, the basilar portion of the occipital bone, as well as the apex of the pars petrosa. These three osseous portions encompass the foramen lacerum. When the tumour tissue grows up through the foramen lacerum it reaches the internal base of the skull in the middle cranial fossa (Fig. 4) with intimate relation to the cavernous sinus and the cranial nerves situated in this place, that is III, IV, V, and VI (Fig. 5). Pressure or direct penetration into the nerves will then bring about increasingly severe lesions particularly of the abducens

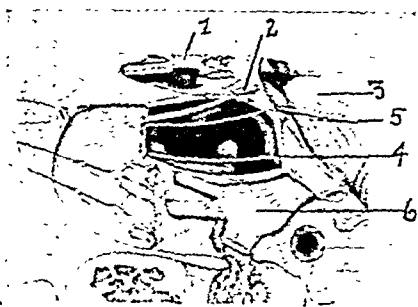


FIG. 5.

Left middle cranial fossa with the cavernous sinus open, seen from the side. Superiorly the optic chiasma (1) and the third nerve (2), under which the thin fourth nerve (5) running along the mesencephalon (3). Further the sixth nerve (4) and the Gasserian ganglion (6) with the ramifications (after *Testut*).

and the trigeminus, resulting in eye muscle paralyses and varying degrees of trigeminal neuralgias or disturbances of sensibility, and possibly paralysis of the muscles of mastication (Figs. 6 and 9).



FIG. 6.

Internal base of the skull of a female, aged 29 years (case 141), presenting a large tumour invasion through the body of the sphenoid bone (destruction of sella and pituitary) and left cavernous sinus from a nasopharyngeal reticulum-cell sarcoma. Clinically there was found left ophthalmoplegia and amaurosis.

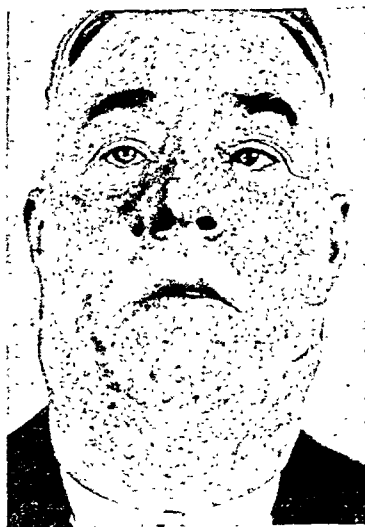


FIG. 7.

Male, aged 56 years (case 81), with left paralysis of the sixth cranial nerve (of one month's duration) and bilateral cervical glands from squamous cell carcinomas on left Eustachian tube.



FIG. 8.

Female, aged 40 years (case 83) with right paralysis of the third cranial nerve (of two month's duration). No metastatic cervical glands. Destruction of the base of the skull verified by X-rays. In the nasopharynx a reticulum cell sarcoma proceeding from right tube.



FIG. 9.

Boy, aged 5 years (case 84) with right total ophthalmoplegia (developed through two months) and nasal stenosis (of five month's duration) as well as right cervical glands (of one year's duration) from malignant nasopharyngeal tumour, presumably reticulum-cell sarcoma.

If the tumour grows upwards and forwards the visual pathway will be injured, and this will result in visual disturbances in the forms of impairment of vision, defects of the field of vision, and different ophthalmoscopical findings, such as choked disc or atrophy.

In cases where the tumour penetrates through the superior orbital fissure there will occur exophthalmos (Fig. 10), which may, however,



FIG. 10.

Male, aged 24 years (case 30) with excessive left exophthalmos and chemosis developed in the course of two months by a reticulum cell sarcoma from the nasopharynx invading the orbit. Cervical gland metastasis the size of a hazel nut (left).

also be due to orbital invasion through the inferior orbital fissure through the parapharyngeal space.

The simultaneous occurrence of ophthalmoplegia, optic tract lesion with amaurosis, and trigeminal neuralgia has been described by Jacod as *syndrome carrefour petrosphénoïdale* and is regarded as pathognomonic of malignant nasopharyngeal tumours.

Penetration of the tumour into the parapharyngeal space in the direction towards the jugular foramen and the hypoglossal canal will bring about different radial symptoms from the cranial nerves running in this area, i.e., IX, X, XI, and XII, with paraesthesias and paralyses of pharynx, palate, larynx, and tongue.

The truncus sympatheticus can be injured partly in this place



FIG. 11.

Male, aged 42 years (case 69) with ipsilateral (right) *Horner's syndrome* (of six month's duration), paralysis of the sixth cranial nerve (of two month's duration, and cervical glands (of three month's duration) from reticulum-cell sarcoma proceeding from the tube (presumably).

and partly intracranially (paratriginally), or in its cervical portion with the development of *Horner's syndrome* (Fig. 11).

Paresis of the seventh cranial nerve, with or without attending eighth nerve lesion, may occur by growth of the tumour in the direction towards the porus acousticus internus; but more often the seventh cranial nerve is injured about its point of exit from the facial canal at the stylomastoid foramen.

All these symptoms are at their initial stages due to pressure from the tumour tissue, but later also to irreversible destructions. That is the reason why nervous symptoms are often seen to improve after irradiation at the initial stages.

Since these ophthalmological symptoms may be the first and only symptoms of a nasopharyngeal tumour, it is no wonder that such cases are often mistakenly diagnosed and treated symptomatically for eye muscle paralyses or trigeminal neuralgia for a considerable length of time without the slightest effect.

The rhinological and otological symptoms in connection with malignant nasopharyngeal tumours are, as mentioned already, due to the exophytic, space-filling growth of the tumour, and also to the tendency of the tumour to ulceration and to development

of stenosis of the air passage, partly through the nose and partly through the Eustachian tube.

The *purely rhinological symptoms* are thus: uni- or bilaterally reduced or arrested nasal respiration, as well as nasal discharge and epistaxis.

The influence of the tumour on the Eustachian tube is sometimes due to mechanical compression and sometimes to penetration of the tumour into the tube. The result is in both cases *tubal occlusion*, which may be intermittent at first, but will soon become permanent, and manifests itself by tinnitus, clicking sensation, and impairment of hearing. The tubal occlusion involves a vacuum in the middle ear with pains in the ear and consecutive exudation, which at secondary infection gives a picture resembling that of otitis media.

Unfortunately the nasal and otogenous complaints are *often misjudged*, so that the patients are submitted to *different operations of the skeletal and soft tissues* in the nose, tubal catheterisations, paracenteses, etc. The frequent mistaken diagnoses are probably due to the fact that the primary tumour is often diminutive and consequently difficult to recognise for untrained examiners.

The fourth and last main group of signs and symptoms is that of *enlarged cervical glands*, which are due to the pronounced tendency of the tumour to early development of lymphogenous metastases, which can reach rather considerable degrees even bilaterally, even when the primary tumours are diminutive.

The typical glandular metastases develop posterior to the angle of the jaw, over the mastoid process beneath the sterno-mastoid, whence they later extend distally. This accords well with the

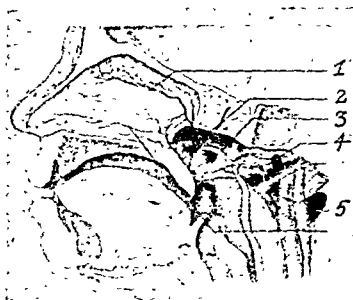


FIG. 12.

Sagittal section of the head seen from within (after Rouvière) with the lymph stems from the nasal septum (1) the lateral wall of the nasopharynx (2) and the tubal ostium (3) to the lateral retropharyngeal lymph node (4) and the superior deep cervical lymph nodes (5).

course of the lymph from the nasopharynx, which passes by the retropharyngeal lymph glands—or direct—to the superior group of the deep cervical lymph glands (Fig. 12). Clinically there occurs (Figs. 13, 14, 15) a steadily increasing, indolent node, whose consistency and fixation to the surrounding tissue depend on the type of the primary tumour, but is generally, however, rather firm and fixed deeply.



FIG. 13.

Male, aged 70 years (case 376) with right cervical gland metastasis in the place of election (developed through nine months) from squamous cell carcinoma in the nasopharynx.



FIG. 14.

Male, aged 50 years (case 1) with bilateral paralysis of the sixth cranial nerve and right paralysis of the third nerve. Large bilateral cervical glands (of one year's duration) from squamous cell carcinoma on right nasopharyngeal wall.



FIG. 15.

Boy, aged 10 years (case 205) with large bilateral cervical gland metastases (of 12 month's duration, regarded as tuberculosis lymphomas. Treated in seaside health resort, etc.), from a large lymphocytosarcoma in the nasopharynx.

The *cervical glands* being frequently the only sign of a nasopharyngeal tumour through half or whole years they are often mistakenly diagnosed as lymphomas of tuberculous or other infectious origin and treated by light baths, incisions, light X-rays, etc.

The *total number of mistaken treatments* for all the groups of symptoms amounts to at least one-third of all the patients.

Syptomatology of malignant nasopharyngeal tumours elucidated from own examination of 454 patients

After this more general review of the symptomatology I shall now pass on to a more statistically based account of the different symptoms.

As it is urgently necessary, in cases of malignant nasopharyngeal tumours, to have the diagnoses made both earlier and with greater certainty than is now the case, it is of importance to concentrate particularly on an elucidation of the early symptoms of these tumour forms. To give as clear a picture as possible of the often very polymorphous course of the tumours I have chosen to make an *analysis of the symptomatology at three different stages of the disease*.

The *first analysis* covers the first symptom(s) observed by the

OPHTHALMO-NEUROLOGICAL SYMPTOMS IN WITH MALIGNANT NASOPHARYNGEAL

patient. It occurs on an average 11 months (3 before the exact diagnosis is made.

The *second analysis* covers the initial symptom in a wider sense. At this stage there are found within one group, or different groups are represented. A clear picture is seen on an average 4 or 5 months before diagnosis is made.

The *third analysis* covers the fully developed stage, which corresponds to the point of time at which the patient is referred to a radiological clinic.

The percentage frequency of ophthalmo-neurological symptoms at these three stages appears from *Table I*, in which the frequencies of the three other main groups have also been entered. The stated percentage figures refer to the 454 cases from my M.I.

Table I shows that ophthalmo-neurological symptoms are the first symptom in 16.0 per cent. of the cases, while cervical glands and rhinological symptoms are the first symptom in about one-third of the patients.

TABLE I.

Percentage distribution of the different groups of symptoms at three stages of the disease among malignant nasopharyngeal tumour.

	Ist symptom 11 months before diagnosis is made	In 4 months before diagnosis is made
Ophthalmo-neurological symptoms	16.0	
Rhinological symptoms	30.8	
Otological symptoms	23.3	
Metastatic cervical glands	32.6	

symptoms are rarer, being present in about

as follows: The *ophthalmo-neurological symptoms* have more than doubled in frequency occurring now in 34·5 per cent., whereas the other groups of symptoms present more moderate increases in frequency, each being found in half of the patients. The frequency of monosymptomatic cases has become reduced to comprising one-fourth of the cases only against nine-tenths at the first stage.

At the *third and final stage of the analysis*—by the time the diagnosis is made—there are two facts which are particularly noteworthy: First the *frequency of ophthalmo-neurological symptoms* is rather stationary, being found in a total of two-fifths of the cases (or 38·0 per cent.), and secondly there is a *considerable increase in the frequency of metastatic cervical glands*, which are now present in three-fourths of the patients. Rhinological and otological symptoms, on the other hand, are rather stationary in frequency, occurring in about half of the cases.

These analyses show plainly that when *ophthalmo-neurological symptoms* do occur in cases of malignant nasopharyngeal tumour they manifest themselves at *early stages*. The ophthalmo-neurological cases can be said to have been separated out as a special group already on an average 4 or 5 months before the exact diagnosis is made. In other words, these symptoms do not—as previously believed—occur only at the advanced and hopeless stages of the disease, a fact which must stimulate our interest in a more energetic tracing of these cases.

The fact that the group of metastases in the cervical glands increases so considerably in frequency from the initial to the fully developed symptomatic picture is a serious reminder of the biological activity and tendency to spreading of these tumour forms. This again means that *if these cases can be diagnosed but a few months earlier than is now the case*, then one of the consequences will be that the patients will be submitted to irradiation before the metastases have spread too far. The chance of obtaining a favourable result from treatment with X-rays will then be increased very considerably.

Thus there appear to be various reasons for stimulating the interest in and knowledge of the initial symptomatology of the malignant nasopharyngeal tumours, in particular the ophthalmo-neurological symptoms.

Nature and frequency of ophthalmo-neurological symptoms

After this statement of the place in the symptomatology of the ophthalmo-neurological symptoms at the different stages of the disease, I shall now pass on to a more detailed description of these symptoms.

Be it stated at once that *all forms of cranial nerve lesions are met with*, as might have been expected from the manner of growth of

the tumour, but that there is found a certain systematisation. Thus for instance lesions of eye nerves and/or trigeminus are found in practically all cases. Before entering on a more detailed account I shall just mention an important proportional change in frequency between eye nerve lesions and trigeminus lesions at the three stages of the disease, where analyses have been made.

TABLE II.

Increase in frequency (per cent.) of ophthalmological symptoms compared with that of trigeminus lesions—from the first symptom to the fully developed picture.

	1st symptom 11 months before diag- nosis is made	Initial picture 4 or 5 months before diag- nosis is made	Fully de- veloped picture when the diagnosis is made
Frequency of ophthalmological sympt. (per cent.)	14.4	51.0	75.2
Frequency of trigeminus lesions (per cent.)	71.0	70.0	68.0

It appears plainly from Table II that the purely ophthalmological complaints constitute a small percentage of the first symptoms, while the trigeminus lesions are in great majority. But at the second stage of the analysis (4 or 5 months before the diagnosis is made) the ophthalmological symptoms have increased very considerably in number, being now found in half of all the cases, whereas the percentage of trigeminus lesions has hardly changed. The increase in the frequency of ophthalmological symptoms continues—though less abruptly so—up to the point of time at which the diagnosis is made, comprising by that time 75.2 per cent. of the cases. They are then more frequent than the trigeminus lesions, which remain at a stationary frequency level (about 70 per cent.).

It is evident from this observation not only that ophthalmological symptoms are present to a considerable extent in cases of malignant nasopharyngeal tumour, but also that these symptoms increase in frequency chiefly within the period in which the exact diagnosis has not yet been made, and in which it should thus be possible to have these cases correctly diagnosed by ophthalmological intervention.

I shall now pass on to an account of the nature of the ophthalmoneurological symptoms. The very first symptom—occurring about 10 or 11 months before the diagnosis is made—is in the majority of the cases (71.0 per cent.) trigeminal neuralgia, most often in the

maxillary area, and more frequently isolated than together with either nasal stenosis, metastatic cervical glands or tubal occlusion. The only ophthalmological symptom at this stage is abducens paresis, likewise more often monosymptomatic than combined with other symptoms. Half of the ophthalmo-neurological cases of my material were purely monosymptomatic, while in the other half the combination forms were rather equally distributed over metastatic cervical glands, nasal stenosis, and tubal occlusion (about 15 per cent. each).

By the time of the second analysis—4 or 5 months before the diagnosis is made—there has occurred not only a quantitative, but also a qualitative increase in the purely ophthalmological symptoms. A detailed description of the distribution will be given in connection with the findings at the fully developed stage of the disease, because there are found no essential qualitative differences between the ophthalmological symptoms of these two stages. I shall only just mention that the purely monosymptomatic ophthalmo-neurological cases are few (3 per cent.) at this stage, since forms combined with rhinological or otological symptoms and metastatic cervical glands respectively are found in 40 per cent. each of the cases.

Accordingly a patient who at this stage of the disease applies to an ophthalmologist on account of eye muscle paresis, visual disturbances, or exophthalmos, with or without attending trigeminal neuralgia, will present symptoms also from the other main groups mentioned here. When the ophthalmologist is consulted by patients suffering from abducens paresis, impairment of vision, exophthalmos, or Horner's syndrome he should always suspect a malignant nasopharyngeal tumour to be the cause. He can then strengthen or weaken his suspicion partly through palpation for metastatic cervical glands in the typical area, and partly by questioning as to the presence of rhinological or otological symptoms, possibly supplemented by an oto-rhinological special examination. The ophthalmologist can thus, through these simple examinations, contribute decisively to the making of an exact diagnosis some therapeutically valuable months earlier than is generally the case.

Of the 454 patients who displayed the fully developed pathological picture 172, or 38 per cent., presented ophthalmo-neurological symptoms.

The nature of the symptomatology appears from Table III, which shows that 75.2 per cent. of the cases had ophthalmological symptoms, and that two-thirds of these patients presented trigeminal lesion as well. Correspondingly two-thirds of the 68 per cent. with trigeminal lesion presented ophthalmological symptoms as well. Four patients only had neither ophthalmological

symptoms nor trigeminus lesion. It appears from this close connection between ophthalmological symptoms and trigeminus lesion that the tumour tissue injures preferably the area round the cavernous sinus, where—as already mentioned—these cranial nerves run in intimate relation to each other.

TABLE III.

Nature and frequency of ophthalmo-neurological symptoms in 172 patients with malignant nasopharyngeal tumours.

Eye symptoms 129 patients 75·2 per cent.	Alone 40 ptt. 23·4 percent.	Ophthalmoplegia 22 patients
		Ophthalmoplegia + II ... 11 patients
		Ophthalmoplegia + Horner 3 patients
		II-lesion alone... .. 2 patients
		Exophthalmos... .. 2 patients
		With V-lesion 44 patients 25·6 per cent.
		With other cranial nerve-lesion, partly V. 45 patients 26·2 per cent.
V-lesions 117 patients 68·0 per cent.	With eye symptoms 78 patients 45·4 per cent.	
	Without eye symptoms 39 patients 22·6 per cent.	

The cases with ophthalmological symptoms can be divided in three almost equally large groups, namely (1) purely ophthalmological cases, (2) forms combined with trigeminus lesions alone, and (3) forms combined with trigeminus lesions as well as other cranial nerve lesions, most often lesions of the ninth and tenth nerves.

The frequency and nature of the individual ophthalmological symptoms proved to be fairly alike for each of these three groups. The symptoms were of the following nature, mentioned in decreasing order of frequency: Abducens paresis, paresis of the third nerve, visual pathway lesion, paresis of the fourth nerve, Horner's syndrome, and exophthalmos.

All the nerve lesions mentioned here can occur as isolated phenomena; but most often they are found in different forms of combination, for instance, as regards the ophthalmological symptoms, in varying forms of ophthalmoplegia with or without attending visual pathway lesion, Horner's syndrome, or exophthalmos.

A direct impression of the frequency of the different cranial nerve lesions is obtained from Table IV, into which have been entered the 461 single nerve lesions presented by the 172 patients.

TABLE IV.

Nature and frequency of 461 single nerve lesions in 172 patients with malignant nasopharyngeal tumours.

Nerve lesions		VI	III	IV	II	Horner	V	Others
Frequency	Abs.	114	42	29	29	27	119	101
	per cent.	24.0	9.1	6.3	6.3	5.8	25.7	21.8

It appears from this table that eye nerve lesions constitute more than half of the total number of cranial nerve lesions, partly in the form of ophthalmoplegias (39.4 per cent.), partly as visual pathway lesions (6.3 per cent.), and Horner's syndrome (5.8 per cent.). Among the ophthalmoplegias the predominating group is that of paresis of the sixth cranial nerve, which constitutes one-fourth of the total number of nerve lesions, being thus almost equal in frequency to trigeminus lesions, which comprise 25.7 per cent.

Pareses of the third and fourth cranial nerves are of much rarer occurrence. The same is the case with pareses of the other cranial nerves (VII, and IX-XII), which altogether constitute 21.8 per cent. distributed over 7 cranial nerves, chiefly the bulbar nerves.

Of the 461 cranial nerve lesions 6.7 per cent. were *bilateral lesions*, which in practically all cases affected the eye nerves. Thus one-third of the optic tract lesions and one-tenth of the ophthalmoplegias were bilateral.

Thus it is plain to see that *the ophthalmological symptoms play a predominant part in the symptomatology.*

The *ophthalmoplegias* generally manifested themselves as massive paralyses with associated clinical findings. The *visual pathway lesions*, on the other hand, presented various degrees of severity from a slight impairment of vision to the more frequent total amaurosis with atrophy of the optic nerve or choked disc. Quadrantanopsia or hemianopsia were of rarer occurrence.

Exophthalmos due to penetration of the tumour into the orbit was found in 19 patients with varying degrees of chemosis and disturbances of motility, usually associated with a poor general state.

I shall not go into details with regard to the trigeminus lesions, but only just mention that they consist most frequently in

neuralgias in one or more of the three branches, whereas the motor root is more rarely affected.

As mentioned before, we have to do with *multiple cranial nerve syndromes* in a great number of the cases; but it would be going too far to go into further details regarding the nature of these syndromes. Yet I want to mention, by way of example, one *cranial nerve syndrome with a special pathogenesis, which has not been described before*, and the occurrence of which must be regarded as *pathognomonic of malignant nasopharyngeal tumours*.

This syndrome comprises *ophthalmoplegia* (most often sixth nerve paresis) and *paresis of the twelfth cranial nerve* and in the majority of the cases also trigeminal neuralgia. It was observed in 9 cases, so there is reason to suppose that it was not an accidental combination. The pathogenesis of the ophthalmoplegia and the trigeminal neuralgia is the usual result of tumour invasion round the cavernous sinus. The explanation of the paresis of the twelfth cranial nerve, which was present without lesion of other distal cranial nerves (so that diffuse tumour growth can be left out of account) is obtained from the lymphatic conditions. Metastatic cervical glands were observed in all these cases, and the primary tumour was found to be lateralised more often than usually, so that the lymph ran to the retropharyngeal lymph glands, which lie off the hypoglossal canal, where it compressed the nerve.

This cranial nerve syndrome is thus conditioned partly by the intra-cranial growth of the primary tumour, and partly by lymphogenous metastases, and is therefore pathognomic of a malignant neoplasm in the nasopharynx.

Roentgenography

Before passing on to the final conclusion I shall briefly state some facts regarding the roentgenography and the treatment in cases of malignant nasopharyngeal tumour.

Roentgenography may, through an ordinary lateral picture of the skull, accidentally disclose a tumour in the nasopharynx, since such a tumour will cause a characteristic deformation of the contour of the wall. More important is, however, the knowledge one gets through a basal picture for an estimate of the extent of a possible osseous destruction. Osseous destruction in the base of the skull with typical localisation in the foramen lacerum area was found in 54.3 per cent. of the ophthalmo-neurological cases. There is an intimate correlation between the roentgenological and the ophthalmo-neurological findings. It may seem an extraordinary thing that nearly half of the ophthalmo-neurological cases presented no osseous destructions, but this is due partly to the fact that the nerve lesions were exocranial, and partly to the fact that the primary tumour can invade the base of the skull through the already existing foramina.

Treatment

The treatment of malignant nasopharyngeal tumours is now purely radiological, as there is contraindication for operative treatment (both from a technical and from a biological point of view) of such deep-seated and invading tumour forms. Fortunately such tumours are generally rather radiosensitive on account of their biologically immature character.

A *strong, external, protracted, fractional X-ray or telerradium treatment* is given, which on rare occasions is supplemented by a local radium or X-ray treatment. The X-ray treatment is given in anterior and lateral fields (Fig. 16) in doses of from 4,000 to 10,000 r.



FIG. 16.

A female, aged 41 years, with characteristic pigmentation of skin portals (lateral and anterior) by the end of an X-ray treatment (9000 r skin doses). Clinically there was found a malignant nasopharyngeal tumour with extensive destruction in the base of the skull, paralysis of the sixth nerve, and *Horner's syndrome*; no metastatic cervical glands. Symptom-free through three years of observation (case 103).

through from 3 to 8 weeks. This treatment can *secure a lasting freedom from symptoms* (over 5 years), but whether it does so depends to a certain extent on the histopathological type of the primary tumour and on the extent of the disease. The radio-sensitive reticulum-cell sarcomas, for instance, show a "five year cure rate" of nearly 40 per cent., while the corresponding figure for the total number of cases amounts to 22.2 per cent.

The *prognosis* is reduced for higher degrees of intracranial invasion; but it should be emphasised that even in cases of cranial nerve paresis and osseous destruction of the base, where all other treatment is in vain, it is possible to secure a lasting freedom from symptoms (over 5 years) by irradiation.

Four-fifths of the patients experience a primary improvement after irradiation; but the lasting results are confounded by a pronounced tendency to recurrence and generalisation, so accordingly the lethality is very high.

Summary and conclusions

Finally I shall sum up what I have said about nasopharyngeal tumours.

The malignant nasopharyngeal tumours are fairly rare tumour forms, but not so rare as they were previously supposed to be. They occur in all age-classes, and are more frequent among men than among women.

The symptomatology, which is very polymorphous and, therefore, to a great extent gives occasion to mistaken diagnoses and wrong treatment can be divided schematically in four groups: (1) Ophthlmo-neurological symptoms due to penetration of the tumour into the base of the skull and the parapharyngeal space, (2) local rhinological, (3) otological symptoms due to the exophytic growth of the primary tumour, and finally (4) metastatic cervical glands, which are typically localised and early developed.

A fractional analysis of the symptomatology at different stages of the disease showed that the ophthlmo-neurological symptoms occur both earlier and to a greater extent than is generally known, and that the ophthalmological symptoms predominate, chiefly as ophthalmoplegias with or without attending trigeminal neuralgias.

Patients suffering from unrecognised, malignant, nasopharyngeal tumours will therefore often apply first to an ophthalmologist on account of ophthalmoplegia or impairment of vision. The ophthalmologist can then do good service in making the exact diagnosis. To do so he must be on his guard with all ophthalmoplegias and visual impairments, etc., of unknown aetiology. The suspicion of a malignant nasopharyngeal tumour is strengthened or weakened partly through palpation for metastatic cervical glands in the typical area posterior to the angle of the mandible, and partly through oto-rhinological special examinations.

If these lines are followed a great number of otherwise mistakenly diagnosed cases will become recognised several important months earlier than is generally the case, and the adequate irradiation treatment will then be instituted at an earlier and, for the results of treatment, more favourable point of time.

The effect of modern intensified irradiation, which is often surprising in the cases of these immature tumour forms, makes the proper background for a more extensive ophthalmo-neurological diagnostication along the lines indicated here, which, when followed, will benefit the patients and reflect great credit on the doctors.

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SOME RESEARCHES ON THE RESPIRATION OF THE CORNEA IN ALBINO RATS*†

BY

A. BAKKER

GRONINGEN

Introduction

IN studying literature dealing with the respiration of the cornea, we get the impression that our knowledge concerning this subject is rather defective. At any rate, we may say that the question whether the cornea uses the oxygen of the surrounding air is still far from settled.

Duke-Elder¹ assumes that the cornea possesses a respiratory mechanism, whereby gases are actively transpired through the agency of the epithelium and the endothelium.

Bullot², arguing on the grounds that in his experiments the endothelium of rabbits' eyes dies when the surrounding air is replaced by a mixture of 1 part air and 14 parts hydrogen, is of the opinion that the endothelium needs the oxygen from the surrounding air. This investigator placed the entire enucleated eyeball of the rabbit, with its epithelium scraped off, in an atmosphere of moist air at 35°C. and observed that, after 15 hours, the endothelium was still living over the entire surface. When, however, the surrounding air was sufficiently rarefied with hydrogen (*e.g.*, 1/15) it was established that after the same length of time all the endothelium cells were dead.

* From the Histological Institute, University Groningen, Director: Professor J. de Haan, M.D. † Received for publication, December 7, 1946.

These facts certainly suggested the possibility that the endothelium uses the oxygen from the atmosphere, but as the experimental circumstances were far from physiological (the eyeballs being enucleated and the epithelium scraped off), these investigations do not determine whether also in the living animal the oxygen passes through the cornea from the air towards the anterior chamber.

Fischer³ has carried out some experiments with the object of examining the respiration of the cornea. All his experiments were performed on rabbits. He constructed a small glass-bell, which was placed upon the luxated eyes of the animals. Thus by means of small tubes furnished with stopcocks, he could fill up the gas-chamber of the apparatus with a definite gas, e.g., carbon dioxide or hydrogen with oxygen. At certain intervals gas-samples were taken out, in order to determine the consumption of oxygen or the production of carbon dioxide. His collective results were shown in a table. He compared among other things, for example, the respiration of a normal cornea with that of a cornea deprived of its layers of epithelium and endothelium. As to the CO_2 -production and the O_2 -consumption practically no difference could be noticed after one hour between the normal and the abnormal cornea; whereas after *four* hours the CO_2 -production was the same for both corneae, the O_2 -consumption, however, of the normal cornea was about 60 per cent. greater than the consumption of the abnormal cornea. So his results showed inexplicable irregularities. From these indeterminate results Fischer derives far-reaching conclusions. This investigator assumes that the cornea forms a barrier to the permeation of O_2 and CO_2 only in one direction, the oxygen being able to travel only backwards through the cornea from the air towards the anterior chamber, the carbon dioxide being able to pass the cornea only in the opposite direction. When Fischer filled up the glass-bell with 100 per cent. CO_2 , he observed that this had a deleterious effect on the cornea within one to two hours. The corneae became totally opaque. Taking into account his view that the cornea is permeable for CO_2 only in one direction, it was obvious that he imputed the injurious influence of this gas to the impediment of the normal permeation of CO_2 through the cornea from behind towards the surrounding atmosphere. As this selective permeability of the cornea for CO_2 however, is by no means certain, Fischer's conclusion, based on this property also lacks reasonable ground.

I got the impression that in Fischer's experiment it was not the normal respiration of the cornea which was measured. In the first place, we notice that the glass-bell was compressed air-tight to the eyeball. In doing this, we cannot expect the normal circulation in the blood vessels of the eyeball to go on without any disturbance

and naturally a change in the respiration of the cornea will be caused by any impediment in that circulation. In the second place, we may ask whether Fischer succeeded in measuring only the respiration of the cornea. Was it possible to exclude any influence of the blood in the surrounding limbus-vessels? And thirdly we must remark that the investigations were carried out on luxated eyeballs. Here too we have a factor which interferes with a normal circulation.

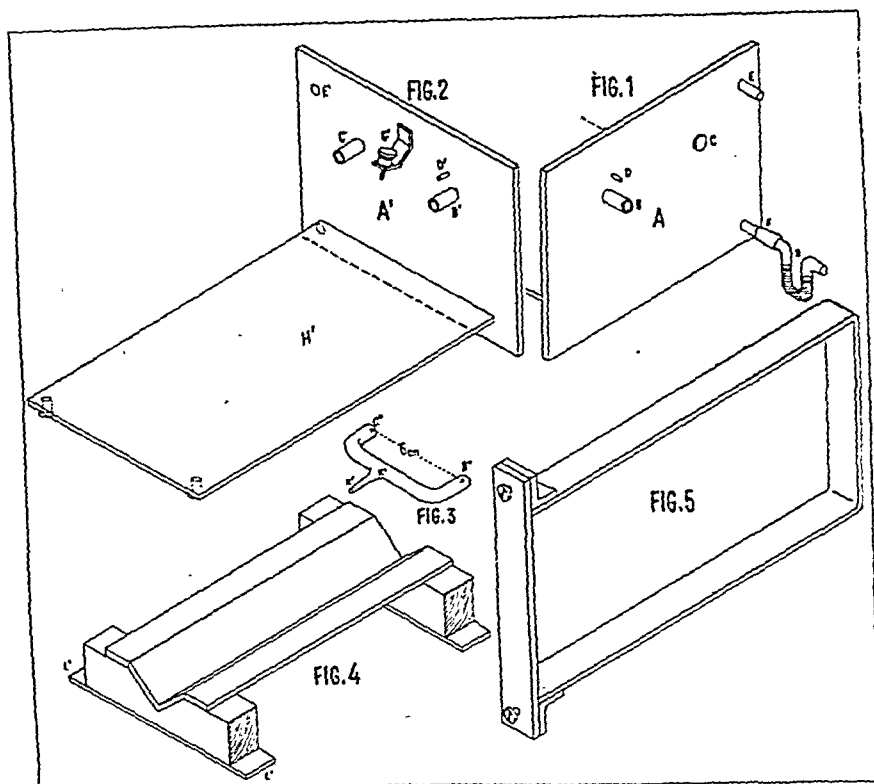
I have therefore thought it worth while to reinvestigate the question of the selective permeability of the cornea. A quite different line of research was started by my experiments.

The anaesthetised experimental animals (albino rats) were put into a closed space in an atmosphere of nitrogen or carbon dioxide or a mixture of these two gases. Of course artificial breathing was necessary to keep the animals alive. The object of these investigations was to examine if any injury of the cornea could be shown under the influence of these gases. We must draw attention to the fact that in my experiments the circulation of the eyes remained under absolutely physiological circumstances (the possible influence of the narcosis we may neglect). The arrangement of my experiments did not allow the measuring of the consumption of oxygen or the production of carbon dioxide, but it was pre-eminently suitable for studying the influence of different gases on normal eyes *in situ*. After an exposure of the eyes to the above mentioned gases for several hours, the animals were killed and the eyes were microscopically examined.

Before stating the results of the experiments we will first give a detailed description of the apparatus used.

A thick walled glass tank (accumulator tank), open at one side and measuring 11 by 14 by 24 cm., was made air-tight by a copper plate (frontplate A in Fig. 1 and A' in Fig. 2) with the aid of a rubber packing. This front-plate was held in position by two u-shaped frames and two strips of copper (one of each is shown in Fig. 5) and thus a closed space was obtained, in which the animals remained throughout the experiments. It is desirable to use a tank made of glass in order to be able to watch the animal in the course of the experiment.

Near the lower edge of the frontplate and parallel to this a horizontal metal plate is soldered (H' Fig. 2). At the back end of this plate are two small knobs on which it rests upon the bottom of the tank, when the frontplate is mounted. The object of this plate is to support a, let us say, small operation table (Fig. 4), on which the animals are tied. This operation table is attached by its metal strips (L') to the horizontal plate with the help of four clips.



The frontplate is pierced by 5 copper tubes. The parts of these tubes inside the tank are indicated in the diagrams by an accented letter, the corresponding end outside the tank by the same letter non-accented. BB' and CC' have a bore of 6 mm. B' and C' are connected by rubber tubing to the trachea cannula. This consists of a right angled curved glass tube (B'' C'' Fig. 3) with a small side-tube K'K' (the trachea cannula proper). B is connected with a small airpump during the course of the experiment. This pump provides a continual air current from B to C. After the animals have been anaesthetised by an injection of a $\frac{1}{2}$ per cent. solution of amytal sodium and after they have been tied on the operation table, the skin in front of the neck is cut through in the median line and, the muscles having been pulled aside, the trachea is brought into view, covered by the deep cervical fascia. The trachea is opened and the trachea cannula is inserted into it. B'' is connected with B' and C'' with C'. Care must be taken not to injure the mucosa or the blood vessels, as the smallest amount of liquid would clog the cannula, causing death by suffocation. If the operation is carefully performed the animals can be kept alive for many hours. By means of the adjusting screw G', resting

upon the glass tube, the trachea cannula K'K' can be inclined in any definite way, so that traction of the trachea is avoided. The volume of air between B'' and C'' is used as a source from which the animal inhales its oxygen and into which it exhales. It may be asked if it would not be simpler to connect the trachea cannula K'K' by means of a lengthening-tube direct with the open air, without using an airpump. This, however, is impossible, as such a narrow cannula would be too long, whereby the resistance, offered to inspiration and to expiration would become too strong.

The small tube DD', with a bore of $1\frac{1}{2}$ mm., is connected at its one end D' by valve tubing to an injection needle and at its other end D with a syringe. Before closing the tank, the syringe, tubing and needle are filled with the anaesthetising solution and the needle is inserted under the skin of the rat. In this way it is possible to give an injection now and then during the course of the experiment, and thus the experimental animals can be kept continually under narcosis for many hours.

By a few sutures the eyelids are prevented from closing. Thus the eyes are fully exposed to the gas under investigation.

A small piece of iron is held against the inner side of the upper tank wall by a magnet outside the tank. By moving the magnet this piece of iron inside the tank can also be moved. This is very

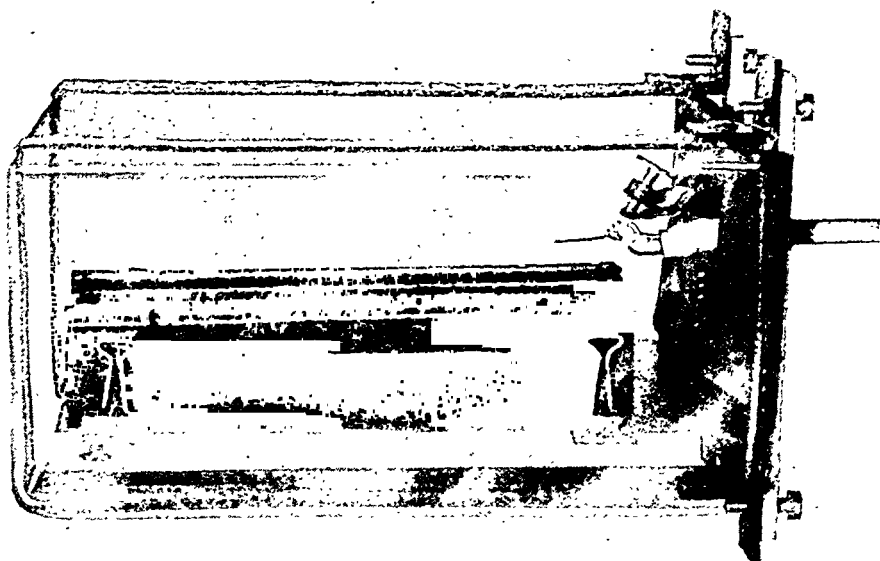


FIG. 6.

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convenient, as it wipes away the condensed water vapour from the inner glass wall, thereby facilitating looking into the tank.

Now the frontplate is brought into its position and the horizontal plate with its operation table and experimental animal is pushed into the tank. The frontplate is closed air-tight by means of the two frames and the airpump is immediately put into action. A photo of the whole apparatus (Fig. 6) elucidates the relation of the different parts in their normal position.

As it is very difficult to drive the air out of a tank by a gas, e.g., nitrogen, and as it is not easy to tell when the moment has arrived that the air is fully replaced by such a gas, I used another method, which allowed the filling of the tank within a short time with the desired gas or gas-mixture in a reliable way.

The copper tube F near the bottom of the tank in the frontplate is connected by rubber tubing to a u-shaped glass tube R. This is the inlet for a 1 per cent. NaCl-solution, previously saturated with the desired gas. The tank is completely filled up with the saline, while the air escapes through the open stopcock in tube E near the upper edge of the frontplate.

When the tank is nearly full of the salt solution, the breathing of the animal is interfered with, as water is little compressible. Therefore we must act very quickly. At the same moment that the tank is fully filled up with the solution, the gas under investigation is let in through tube E, while the salt solution can flow away through tube F. When the water has fallen so far that the level stands in the upper part of both the legs of the u-shaped tube R, the stopcock in E is closed. In this way we have achieved our object of filling up the tank quickly with a definite gas.

We may neglect the influence of the vapour-tension of the water, as diminishing the tension of the gas under investigation.

The operation on the experimental animals and all the further manipulations until the end of the experiment, were performed in a room at a temperature of 37°C.

Description of the results

The albino rats used had an average weight of 180 to 200 grs. They were anaesthetized by a subcutaneous injection of 4 c.c. of a 0.5 per cent. solution of amytal sodium in aqua dest. After about 20 minutes the narcosis generally was complete. Every two to three hours 1 or $\frac{1}{2}$ c.c. of the solution was injected in order to keep the animals under narcosis. The next quantum of the narcotic was generally given when the animals showed some signs of re-awakening, for instance when just perceivable contractions in the legs were visible. It is clear that this made it necessary to watch the animals constantly. A good indicator for judging if the respiration of the animal is going on normally is the pink colour of

the tail. The slightest disturbance in the respiration betrays itself by cyanosis of the tail.

The gas being saturated with water-vapour, the eyes will be protected from desiccation, though lacking the normal blinking of the eyelids.

In the first place my experiments had the object of investigating whether absence of oxygen in the atmosphere would cause any pathological changes in the cornea.

I obtained results similar to Fischer in so far that I could show that normal life of the cornea is possible in the absence of oxygen. After a stay of 12 hours in an atmosphere of pure nitrogen, the corneae remained quite clear and microscopically no injurious influence was found. In fact we could hardly have expected anything else. I only need to point to the fact that a whale can remain under water for some hours, that is in a medium with a very low concentration of oxygen, to understand that, besides the oxygen normally supplied by the blood vessels in the eye, the cornea does not need any other source of oxygen to preserve its integrity. If the normal respiration of the cornea depended on the oxygen content of the surrounding air the eyes would certainly get into a precarious position during sleep, when the eyes are hidden behind the closed eyelids. Altogether we may say that the evidence points to the fact that in normal circumstances the cornea does not use the oxygen from the surrounding atmosphere.

In order to investigate the influence of CO_2 on the cornea, in the first place some experiments were carried out on rats in an atmosphere of pure carbon dioxide. Within half an hour the corneae became totally opaque. Consequently the deleterious effect of CO_2 is obvious. The results of these experiments agree with those of Fischer. From such evidence alone, however, it could not be argued that it was an impediment in the normal permeation of CO_2 through the cornea from behind towards the air which caused this injurious effect. On the contrary, the probability was great that the high concentration of CO_2 , being far from physiological, was the direct cause of the death of the corneal tissues.

It is clear that as Fischer's experiments were not carried out under physiological conditions, they did not prove the selective permeability of the normal cornea. Moreover, such a selective permeability in other animal membranes is still quite unsettled, *e.g.*, Wertheim's experiments showing the permeability of NaCl through the frog's skin only in one direction could not be reproduced.

Even my own experiments cannot give a definite answer to the question whether a selective permeability of the cornea exists or not. Yet they have made this assumption very improbable. The following experiments show that a stay of several hours in a CO_2

concentration, higher than any normal CO_2 concentration in the body (8 per cent.), does not cause any pathological changes in the cornea.

Assuming that carbon dioxide normally diffuses away through the cornea from the anterior chamber towards the surrounding atmosphere, and assuming with Kronfeld⁴ that the CO_2 -tension in the aqueous humour lies between the CO_2 -tension of the venous blood and the arterial blood, we may be certain that a concentration of 8 per cent. of carbon dioxide outside the eye is high enough to impede the eventual permeation of CO_2 through the cornea.

This concentration, being not far above the normal CO_2 -tension in the tissues, has the advantage of being almost physiological and it is not to be expected that it will have a direct noxious influence upon the cornea.

Some introductory experiments showed us that after two hours in an atmosphere of 8 per cent. of CO_2 and 92 per cent. of nitrogen the corneae remained fully transparent. When we compare this with the injurious effect of 100 per cent. of CO_2 , causing the death of the corneal tissues within half an hour, we notice a striking difference. It goes without saying that the conception that the deleterious effect of CO_2 is caused by an impediment of the permeation of CO_2 through the cornea towards the exterior air, must now be rejected.

Repeated observations under the same circumstances as to the CO_2 -concentration (8 per cent.), but after more prolonged times (10 hours and more) have shown that no pathological changes were caused in the cornea. Histological investigation showed us that the corneae were normal in every respect. In one section sometimes 16 karyokinetic figures were seen in the deep layer of the corneal epithelium. This means that in such a cornea a few thousands of cell divisions occurred simultaneously. We could hardly wish for a more convincing proof of the integrity of the cornea. At first I was of the opinion that this large number of cell divisions was abnormally high, and that it was the consequence of an irritating action of the CO_2 -gas.⁵ However, on counting the cell divisions in a cornea of normal rats of the same age, the number of mitoses found here, was not considerably lower than in the cornea of the experimental animal. Thus we may conclude that also in this respect the cornea of the experimental animals does not show any abnormality.

Since my experiments have shown that it is impossible to accept a noxious effect of one or other factor causing an impediment to the hypothetical normal diffusion of the carbon dioxide through the cornea towards the exterior air, we must give an explanation of the deleterious influence of *high* concentrations of this gas (e.g., 100 per cent. of CO_2). I believe that we must assume a direct

noxious influence of the CO_2 causing rapidly the death of the whole cornea. In other words, I am of the opinion that carbon dioxide is able to permeate through the cornea from the surrounding atmosphere towards the anterior chamber, the possibility of which Fischer denies.

Summary

In this paper I have given a description of some experiments which had the object of studying the influence of various concentrations of CO_2 and the absence of oxygen in the surrounding atmosphere on the cornea of anaesthetised albino rats. With the help of artificial breathing it was possible to close up the animals in a tank filled up with one kind of gas or another, and in this way to expose the eyes of the experimental animals under otherwise normal conditions to the gas under investigation. It could be determined that the so-called selective permeation of carbon dioxide through the cornea is not probable. Absence of oxygen in the surrounding atmosphere does not inhibit normal life of the cornea. The corneae remained transparent for many hours in an atmosphere of 8 per cent. of carbon dioxide and 92 per cent. of nitrogen. Special attention was paid to possible pathological changes in the corneae after the experiments were finished.

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CONTRIBUTIONS TO TOTAL BLEPHAROPLASTY*†

BY

IDA CZUKRÁSZ

DEBRECEN, HUNGARY

THE substitutions of deficiency of the whole lid is one of the great problems in plastic ophthalmic surgery and more so, if the upper lid is absent. There have been several methods published, recommended and employed. Here I want to show three different methods of restoration—better to say substitute—for upper lid loss, providing suitable outer and inner layer at the same time.

CASE 1 (Fig. 1). The left upper lid of a lady aged 60 years was

* Received for publication, November 15, 1946.

† Report from the University Eye Clinic, Debrecen. Leader: Professor Kettesy.

infiltrated with carcinomatous tissue. The tumour was the size of a green almond with ulcerated surface and of very interesting origin. Several months before the plastic operation was done she had been twice operated on for multiple chalazia. The tumour developed possibly out of chronic inflammation of the Meibomian glands. This was shown by microscopic sections. The pathologist's diagnosis was carcinoma alveolare.

After the extirpation of the tumour the whole upper lid was lost, only a one and half cm. broad skin strip was left beneath the



FIG. 1.

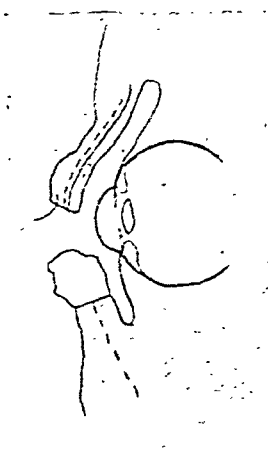


FIG 2.

eyebrow. The bulbar conjunctiva was intact with 3 mm. of upper fornix. Here we decided to employ Hughes's method, but reversed. He rebuilds the lower lid out of the posterior layer of the upper lid, and of the skin and even of the peri-orbital parts of the face. The procedure is based on Landolt's and Kuhnt's ideas, who actually establish a total blepharorrhaphy between the lid-stump and the split normal lid. The two layers are separately united and in a different level. (Figs. 2, 2a, 2b).

Fig. 3 shows the state after the blepharorrhaphy.

Nine months later a new palpebral fissure was opened at the the desired height (Fig. 4). We hope to get in another few months a fairly good function also.

CASE 2. An old lady aged 71 years lost the left upper lid, a basal-celled carcinoma infiltrated one third of the width and the entire length of the lid. The surface was badly ulcerated.

In this case we chose Blasković's II method. The tumourous eyelid was excised through healthy tissue. After that we mobilised the remaining conjunctiva of the eyeball and putting 3-4

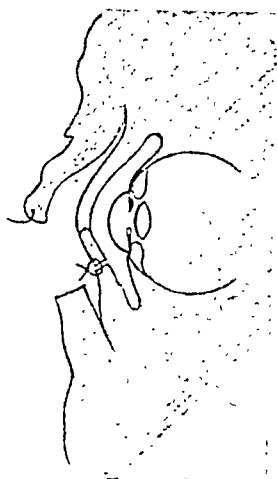


FIG. 2a.

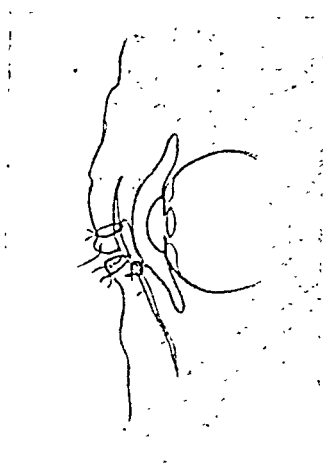


FIG. 2b.

simple sutures in its border, sew it to the skin of the lower lid, at the level of the orbital margin (Fig. 5). Then, with a bridge-flap of 1.0–1.5 cm. breadth, outlined parallel with the eyebrow, the

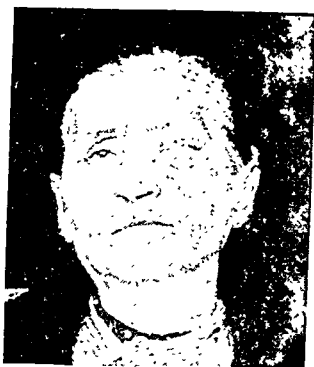


FIG. 3.



FIG. 4.

anterior layer of the future lid margin was performed (Fig. 6). The secondary large defect was covered with a Kettesy graft. Figs. 7 and 8 show the result after the operation.

CASE 3 is of particular interest. I have shown it already at the Oxford Ophthalmic Congress in the year 1938, then speaking generally of the arched plasty. It occurs very seldom, one has to restore both lids, and to be able at the same time to save the eye

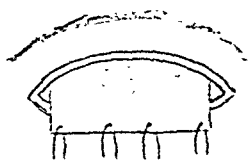


FIG. 5.

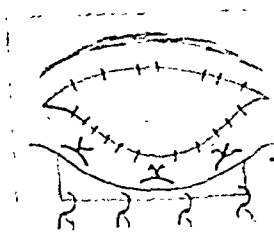


FIG. 6.

too. That happened in our case, a woman aged 57 years (Fig. 9) had an enormous carcinoma partly cicatrised in consequence of other treatment applied elsewhere, partly progressive of the baso-cellular type. The eyeball was intact with sight :1, so we decided to save the bulb. We removed of the total upper and lower lid, inner and outer canthus and a piece of the forehead skin. Then we had the problem how to substitute both eyelids with both

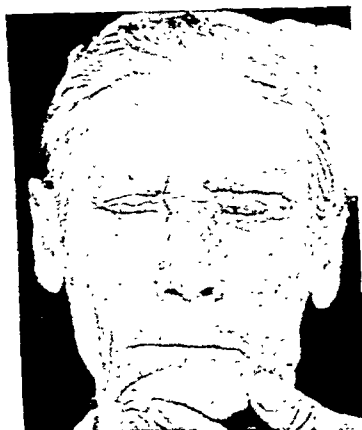


FIG. 7.



FIG. 8.

layers. Fortunately some bulbar conjunctiva was left, and we thought the posterior layer could be done by that. We covered the huge defect with a single sliding flap after Blaskovics, known also as Hungarian plastic (Fig. 10). This extended from the inner canthus to the auricular lobe. The flap was perfectly living, but we were not sure whether the conjunctival sac would develop that we wanted so badly. Eight months later our patient returned, and then one could feel the moving eyeball, and above this a cystoid growth which had developed underneath the flap. Naturally this was a good sign because the walnut sized tumour was a cyst in that

the conjunctival remnants met above the cornea. So we could hope to find, by preparing with a horizontal cut an eyelid fissure, the cornea unaltered. Moreover we were intending to form the eyelids having there already some conjunctival tissue.



FIG. 9.



FIG. 10.



FIG. 11.

When the eyelid fissure was actually done we found the eyeball intact, sight unaltered. Later on from the viewpoint of cosmetics we made an attempt at tattooing an eyebrow (Fig. 11). We intended also to make some kind of ptosis operation, but we lost our patient by the war.

Summary

These cases above represent three different methods of substituting the total lack of eyelids. Applying these methods we never failed. The first procedure is Hughes's method, that is equally fit to restore the upper and lower lid. Blaskovic's II operation is suitable when the whole upper lid is missing. The Hungarian plastic generally is used only for substituting the lower lid but in cases when both lids are lacking we recommend the sliding flap, moreover if we hope to save the bulb, being able to replace at once the upper and lower lid with one arched plasty.

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 ANNOTATIONS

On Forewords

It is becoming increasingly common nowadays to find new medical textbooks issued from the press with a foreword which, in most instances, has been contributed by some physician or surgeon of outstanding personality. It is one of the penalties attaching to the exalted position they have reached in their profession.

Johnson's Dictionary makes no mention of the word, but the Shorter Oxford Dictionary dates it from the year 1842 and defines it as "a word said before something else, hence a preface." But it is not the recognised preface in professional books, for this is always written by the author. The word is not a particularly pleasing one, and we might say, as Mrs. Blimber said of the name of Mr. Glubb, the old man who drew the bath chair in which little Paul Dombey was dragged on the sands of Brighton, that it is unclassical to a degree. If a really classical substitute is desired we can think of no better word than *prolegomenon*. *Prelude*, *proem* and *introduction* are quite unsuitable and we do not much care for *preamble* or *prologue* in this connexion.

Altogether we fear that the foreword must stand, for we do not see what better word could be chosen. When we began this note we toyed with the phrase "Grace before Meat"; but have decided that it is hardly suitable and the shade of Charles Lamb might be offended; and the proposition might be maintained that in this case the grace should be placed after the meat and not before it. We refrain from drawing a parallel between the foreword and *placenta praevia* or between an *epilogue* and the after birth.

The Surgical Team

IN the modern operating theatre perfection can only be obtained by a good team, every member of which from the 'chief' to the theatre attendant must know when and how to play his part accurately, carefully and with perfect timing in order that the patient may have an operation performed efficiently and expeditely. Disharmony is inevitable when despite a 'chief' whose operating ability is of a high order his assistant is clumsy or unable to anticipate the next move ahead, or the nurse in charge of instruments delays in passing these, or the theatre orderlies are noisy and heavy-handed in the transfer of the patient to and from the table. The team drill of an eye operation should be so well understood by all those taking part that the surgeon should have no cause to speak except for a preliminary word of encouragement to a patient under a local anaesthetic and a few directions to him if need be.

In the large teaching hospitals in London and certain provincial towns where the smooth running of a surgical team should be exemplary to all those who visit such centres of instruction, this ideal is rarely maintained for long. Every six months a trained assistant leaves and his place is taken by another inexperienced in the ways of his surgical chief. The more frequent changes in nursing staff upset the team work even more than the change of assistant. It seems that most matrons consider it necessary for all nurses to 'go through' the theatres and many who may be good and efficient in the wards have no aptitude for theatre work, and indeed some are a menace to the safe conduct of an operation and to the proper care of instruments. This circulation of untrained medical and nursing assistants is considered necessary for teaching purposes.

In the Army field hospitals during a busy intake of casualties it sometimes happened that a key member of a trained team would be taken away. It was in vain that one argued with matrons that whilst such changes may have had an educative justification in peace time that under active service conditions such a move was uneconomical in time and efficiency. The parallel illustration of taking a gunner out of a tank crew and replacing him with a pioneer was not appreciated.

Is it always necessary to train the embryo eye surgeon to be a good assistant before he is capable of operating and is it desirable for all nurses to receive a smattering of theatre training for a time quite inadequate to do any good?

What is the ideal? In a large teaching hospital we think that a surgical team should be carefully selected, drilled and kept together. It is desirable also to train reserves who can fill a vacancy in the event of illness, holidays and other eventualities. These reserves could be composed of the resident in training to take over the duties

of his predecessor and similarly a nurse understudying the 'charge nurse.' Only with such a team is it possible to demonstrate the smooth sequence of operative steps efficiently and expeditely.

THE OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (British Medical Association)*

Annual Meeting

THE sixth annual general and scientific meeting of the Ophthalmological Society of Australia (British Medical Association) was held at the Royal Australasian College of Surgeons, Spring Street, Melbourne, on October 23, 1946. The President, Dr. J. Ringland Anderson, occupied the chair. Those present included three members from Queensland, ten from New South Wales, thirty-one from Victoria, two from South Australia and two from Western Australia.

President's Address

The President, Dr. J. Ringland Anderson extended a welcome to such guests as had been able to attend, but particularly to Air-Commodore P. C. Livingston, C.B.E., A.F.C., F.R.C.S., R.A.F., who had flown from Singapore to attend the meeting. A tribute was paid to the Ophthalmological Society of the United Kingdom. Blindness was considered as our common enemy. Various ways in which it is most frequent were dealt with as if blindness was an anthropomorphic enemy. These ways included congenital cataract, tumour formation and the effects of trauma. Defects in our defence against this enemy were discussed, such as those that occur in our training of the recruits and in our vision of the enemy. It was emphasised that we must see not simply the disease but the individual as a whole, and he in his social and his cosmic setting if we are to obtain a complete view of our patient's problems. The slow evolution of the spirit of medicine through the ages was briefly traced. It was suggested that bureaucratic control could unwittingly interfere with this spirit and blindness ensue. The victims of blindness and their rehabilitation were considered. It was decided that many of them saw in life what many of us who are "sighted" fail to see. Reference was made to what was called real blindness and to the influence of atomic warfare.

Unveiling of memorial to the late Major Z. Schwartz

A bronze tablet, in perpetuation of the memory of the late Dr. Z. Schwartz, who was killed in action on April 10, 1941, was unveiled. Doctors A. S. Anderson and T. a'B. Travers spoke with

* Received for publication, January 7, 1944.

deep appreciation of the colleague the Society had lost and of his contribution to the establishment of the Medical Eye Service of Victoria. It is intended to hang the tablet in the premises of the Medical Eye Service.

The Relationship between Night Vision Testing and Night Vision Training in Aerial Warfare

Air-Commodore P. C. Livingston described the early experiments carried out by the R.A.F. in night vision and its influence on flying. He traced the gradual development of the rotating hexagon and discussed its virtues and its limitations. The term "night visual capacity" was shown to be very useful. The factors which tend adversely to affect this capacity were considered. Particular attention was paid to anoxia. Its effects on the hexagon test and on the erratic involuntary ocular movements were shown by slides. The night vision screen test was described. Fields were shown to exhibit the results of anoxia, vitamin A deficiency, retro-bulbar neuritis and diabetes. The probable trends of future development were discussed.

The Cure of Heterophoria in Air Crew—Its Clinical and Psychological Significance

Air-Commodore P. C. Livingston, R.A.F. described the changed outlook on the influence of heterophoria on flying. Reference was made to the origin of certain tests and to their usefulness. Their susceptibility to psychological and neuro-muscular influences was considered. The value of orthoptics both before flying training and as a means of rehabilitation was emphasised. Tribute was paid to the work of English orthoptists during the war. Cases of special interest were received and the results of their orthoptic training were described. Speculation was made regarding future developments.

The rôle of Orthoptic treatment

Miss Diana Mann (Melbourne) read a paper which attempted to show types of squint and other defects suitable for orthoptic training. It was assumed that treatment was not justified when results were slow or uncertain. Treatment was most useful for accommodative squints, certain occasional squints, and as an adjunct to operation for certain symptoms of eyestrain which did not respond to correction of refractive error. In such cases lack of voluntary control of convergence was more significant than muscle imbalance. Success always depended on the patient's mental and physical health and powers of concentration.

Concussion Glaucoma

Dr. Arthur D'Ombrian (Sydney) attempted to clarify the views expressed in an earlier paper upon traumatic glaucoma.

The nature of the lesion was defined as a true chronic non-congestive glaucoma, and attention was drawn to the delay in the onset of symptoms, as this was evidence that no gross visual upset occurred at the time of injury. Three types of trauma occurred in these cases, namely:—blows upon the eye, injury to the skull and heavy falls.

These injuries could be regarded as forms of concussion and the resultant chronic glaucoma was suggested to be one of the several possible results of concussion of the ocular bulb. Additional case histories were described.

In conclusion, the hypothesis was put forward that these cases were indicative of a condition which might be named "concussion glaucoma," and that the mechanism of their production was that of a sclerosing lesion initiated by trauma, a fibrosis of the ciliary region or of the venous-capillary bed. This fibrosis is progressive and goes on to the production of the ocular oedema known as glaucoma.

Such a hypothesis explained the remoteness of the history of injury in many of the cases. The lesson was not too hastily to diagnose every case of chronic glaucoma in an eye as a bilateral disease, for it might turn out to be a monocular lesion secondary to a concussional trauma.

A Survey of Retrobulbar Neuritis in Prisoners of War

Dr. S. R. Gerstman, as an ophthalmologist at 115th Australian General Hospital, Heidleberg, at Stonnington Red Cross Rehabilitation Centre for blinded servicemen, was associated with many cases of what was now called retrobulbar neuritis due to avitaminosis. Results of examination were described and vision of all cases at both institutions were summarised. Fundi and fields were discussed and treatment and prognosis were described. Dr. Gerstman gave details of rehabilitation and mentioned the percentage of disability and pensions. Possible liaison with St. Dunstan's of England was also suggested.

Dr. R. B. Maynard who had been a prisoner in Changi camp described the microscopic findings of the ocular tissues he had brought back. Unfortunately his difficulties in preserving the material were such that the detail was not very good. He considered that the changes were such as have been attributed to beri-beri.

Retrobulbar Neuritis and deficiency disease

Dr. Clifford S. Colvin (N.S.W.) gave a short survey of men who had returned from imprisonment in the Halmaheras, Ambon, and Borneo. These men were first seen at Morotai and others came from Changi and Kranji camps where Majors Claffey and Orr had looked after them. The men examined comprised English, Dutch, Australian and Indian troops and a few Dutch women.

It was suggested that possibly an unknown factor in addition to vitamin deficiency was to be looked for in this type of case. Its effects on other parts of the eye and ocular functions were also considered. Dr. Colvin said that one definite case of vitamin A deficiency was encountered responding well to treatment. Some men who lived under similar conditions showed no defects or symptoms, yet their diet must have been similar to those who did get symptoms. Men of all ages were affected by the disease and apparently age was not significant. Some men who said they had blurred vision a year or two previously when examined had normal vision of 6/5.

Dutch civilians, men and women, who had been examined, showed both normal and defective vision. The few children who had been examined showed no defective vision.

Men from Ambon, Dr. Colvin continued, who had no specialist treatment, showed some who were affected, mildly and severely and others who showed no effects at all. The rice ration in Ambon was about half that in Singapore. The incidence of defective vision seemed lower in men who had returned from Kuching in Borneo.

The general symptoms were usually numbness of the feet—feeling of pins and needles—aching in toes, feet and arches, with difficulty in walking. Back of the calf tender and knee joints painful—often associated with conjunctivitis and “glare”—vision then became blurred, oedema of legs and body occurred later, and most patients described themselves as having “beri-beri.”

Inherited Retinal detachment

Dr. J. Bruce Hamilton (Hobart) presented a pedigree of inherited juvenile retinal detachment. A total of ten patients, with 20 eyes, of the pedigree were examined. Six patients with a total of ten eyes were refracted and the whole were refracted and the whole were hypermetropic. Of the total ten patients examined five had retinal detachment involving seven eyes. Of these seven eyes, three had pseudo-glioma, one retinitis proliferans, and one a detachment which reattached itself. A further two of the ten patients examined had unilateral complicated cataracts, and loss of projection with undoubted detachments behind them. Dr. Hamilton said it was therefore safe to presume that seven patients and nine eyes were involved with detachments in the pedigree. Of the remaining eleven eyes, one had a retinal cyst with peripheral choroido-retinitis, and three eyes had peripheral choroido-retinitis without retinal cysts. One eye of the twenty examined had aniridia.

Dr. Hamilton then discussed the inheritance of polycystic disease of the kidneys, liver and pancreas, and the inheritance of congenital cystic disease of the lungs. He was able to show a film of one patient from his pedigree with retinal detachment in both eyes and pseudo-glioma who had congenital cystic disease of the lungs in an

advanced state. He asked if members of the Society would enlighten him as to the relationship between congenital cystic disease of the lungs and inherited juvenile retinal detachment. He felt that they were both due to generalised congenital cystic disease involving the eyes and the lungs for at least two reasons:—

(a) that the retinal cysts become infected and appeared as pseudoglioma just as the lung cysts became infected and bronchiectasis ensued and

(b) the fact that like lung cysts, retinal cysts ruptured and the detachments re-attached themselves.

Angeoid streaks of the Retina

Dr. J. D. Maude (Sydney) gave a description of cases of angeoid streaks of the retina with pseudo-zanthoma elasticum. Dr. Maude also gave a short review of contributed cases by other members, and discussed the literature.

Pigmentary abnormality in children congenitally deaf following maternal German Measles

Dr. E. O. Marks (Brisbane) said that though incidence of congenital cataract did not rise in Brisbane as in Sydney, Melbourne and Adelaide, there were serious "epidemics" of deafness and cardiac defects. Half the deaf children born in the years 1937, 1938 and 1941, however, showed retinal pigmentation while those born in other years were normal. The appearance resembled a typical form of retinitis pigmentosa.

Intracapsular cataract extraction, its most serious complication

Dr. F. Gregory Roberts (Sydney) said that the condition described consisted of a progressive retraction upwards of the iris following a technically perfect operation. It was not connected with the loss of vitreous at the operation or of any subsequent burst through of vitreous. The types of patient in which the condition occurred were described and treatment suggested.

The Future of Ophthalmology

Dr. K. O'Day (Melbourne) said that because of lack of teaching of the subject, ophthalmology in Australia had always rested on a very insecure foundation. Examinations held in the past had not been very satisfactory, and the facilities provided for training were quite inadequate. Histology should be made a living subject, and a library of slides for the teaching of histology and embryology should be available. Physiology should provide a sound introduction to clinical ophthalmology. The clinical aspect of the subject should be

taught from the patient and not from a book. It must of course rest on a sound basis of pathology. The opportunities available have been neglected in the past. Without a sound basis of pathology, no progress could be made. Adequate direction was essential, said Dr. O'Day, and this could be provided for by a Chair of Ophthalmology at the University.

A clinical meeting was held on Saturday morning, October 26. A series of very interesting cases was demonstrated. Dr. Kevin O'Day demonstrated the rapid method of celloidin embedding and a large number of slides of ocular tissues of Australian animals. Dr. Ringland Anderson demonstrated the projection and the polaroid methods of investigating diplopia. He emphasised the value of "dividing" diplopia into a component for each eye, and he described the association of palsies of one superior muscle of each eye, of one inferior muscle of each eye and of both superior oblique muscles.

Annual Dinner

The Annual Dinner was held at Menzies Hotel, and was attended by 45 members and the following guests:—Air-Commodore P. C. Livingston, R.A.F., Professor P. MacCallum, Professor Sydney Rubbo, Air-Commodore E. A. Daley.

The President announced that Air-Commodore Livingston had accepted honorary membership of the Society. The announcement was received with applause.

The President offered the congratulations of the Society to Dr. N. McA. Gregg, who had received the Shorney Prize, at its first award, for his work upon "Congenital cataract associated with maternal rubella."

Annual General Meeting

The annual general meeting dealt with general business. Particular attention was given to special problems, including ophthalmic education and the future of ophthalmology.

Dr. Darcy A. Williams (New South Wales) was elected President for the ensuing year.

It is proposed to hold the seventh annual general and scientific meeting at Sydney in the week September 1 to 6, 1947.

BOOK NOTICE

Eye Surgery. By H. B. STALLARD, M.B.E., M.D., F.R.C.S. Pp., 444, 338 illustrations. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall, Limited. 1946. Price, £2 10s.

To have written a book of this scope, with such wealth of detail, and to have drawn the majority of the illustrations himself is an achievement of which any ophthalmologist might be proud. To have written it without access to ophthalmic literature, at sea, in military transports and in camps, with all the distractions incidental to active service makes the achievement still greater. In one way, it also enhances the value of the book, because, instead of being presented with a rehash of the often conflicting views of many authorities, the reader is given the results of one man's observations, and the methods he has used to cope with the many and manifold surgical problems presented by the eye and its adnexa. Were the experiences of such a man narrow and limited, this method of describing it would be of little value; but in the present instance, the author is a man of wide experience—both in civil and military fields, and the results of this are gathered together in the volume under review.

It opens with an introductory chapter, which abounds in useful instructions for the ophthalmic surgeon and his assistant, and deals with matters such as the lay-out of the theatre, care of instruments, discipline of the staff and pre- and post-operative care of patients. This is followed by a chapter on anaesthesia and analgesia, and then come some 130 pages concerned mainly with plastic surgery of the eyelids. In addition to describing operations for the relief of ptosis, ectropion, entropion and other deformities, the author deals in considerable detail with reconstructive surgery for war injuries. This is regarded by many as the proper domain of the plastic surgeon, but a good case is made out for the ophthalmic surgeon doing this work, though he should have an apprenticeship with a plastic unit before attempting it. The same question comes up with regard to dacryocysto-rhinostomy, which some regard as an operation for the rhinologist. Although it would probably be unwise for the ophthalmic surgeon to attempt it without having previously seen the operation, he should be able to carry it out by following the careful description and clear illustrations given in the letterpress.

The remaining chapters of the book deal with the lacrimal apparatus, the extra-ocular muscles, the conjunctiva, cornea, anterior chamber and sclera, the iris, the lens, glaucoma, the retina, choroid and vitreous, traumatic surgery and the orbit. Throughout these, the same meticulous care is observed in the illustration and description of the operations. Each is preceded by

a list of the instruments required, and an account of the method employed for producing anaesthesia or analgesia. Although this entails a good deal of repetition, it has the advantage of giving a complete description of each operation, which is useful for hasty reference. The author never hesitates to incorporate his own views, and is apt to give these at times in a dogmatic fashion—an example being the statement that, in severe cases of squint, operation is done between the ages of 2 and 4. In the reviewer's opinion, such dogmatism is justified when one takes into consideration the purpose of the book; but it might afford a subject for criticism by some readers.

"Eye Surgery" contains much original matter, which has not appeared before in book form—particularly striking examples are afforded by the description of the author's methods of extraction of intra-ocular foreign bodies by the posterior route, of implantation of radon seeds far back on the eye for intra-ocular neoplasms, and of his corneo-scleral stitch in cataract extraction. In describing the extra-capsular method of performing this operation, it is recommended, after applying and closing the capsule forceps, to displace them down, and then to the temporal and nasal sides before removing them. Such a manoeuvre has led at times to loss of vitreous, whereas this catastrophe does not occur if no manipulations are made preliminary to taking the forceps out of the eye once they have grasped the capsule.

The chapter on the orbit contains much useful information, and an excellent description of a modified Krönlein's operation, which, however, would be easier to follow if a diagram of the bony structures were included, showing the lines along which they are divided.

The function of a review of a book is to indicate the characteristics and contents, so that readers can decide whether or not they need to possess it. In the present instance, the characteristics are lucidity of description, meticulous attention to detail, incorporation of valuable material not heretofore obtainable in book form and sincerity, with a certain healthy degree of dogmatism in its writing. As a result, the reader of this volume, given reasonable experience in ophthalmic surgery, should be able to perform any of the operations described in it, even if he has not had the opportunity of seeing them done by others.

For many years a need has been felt in this country for an authoritative English book on ophthalmic operative surgery, and this need has now received ample satisfaction.

We would like to congratulate 'Henry' Stallard on having achieved what might seem the impossible—namely, providing us with corn from Egypt and manna from the desert.

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 of yong gowys. geided soley. and
 not rawe tyll they be reuined.

Deo graas.

CORRESPONDENCE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—

"De Oculis, by Benevenutus Grassus of Jerusalem."

In 1929, the late Casey A. Wood published an English translation of the first printed edition at Ferrara, 1474, A.D., of which Incunable, he gave illustrations of the first, 17th, and last pages; and an illustration of the "Explicit" and last folio of an Old English codex from the Ashmole collection in the Bodleian Library, Oxford.

Wood stated that that was the only English translation he was able to find, apart from his own translation. The old codex he found was a fragment of six pages.

During my investigations for editions of Arabic ophthalmology, last year in the Hunterian Library of the University of Glasgow, I came across the following,

Manuscript 303. Beneventi Graphi (Grassi).

De VSV Oculorum, Opus in Linguam Anglicum translation 68 pages.

Vellum binding; 68 pages well written in single columns, 15 lines each $4\frac{1}{4}$ by $3\frac{3}{8}$ inches; gilt illuminated capitals. The late editor of the Catalogue of the Manuscripts, placed the date of this MMS. in the XV century.

At the forthcoming Glasgow meeting of the Ophthalmological Society of the United Kingdom at the end of March, 1947, the President desires this volume to be displayed, with other rarities, showing editions recording early discoveries; and the Librarian, W. R. Cunningham, Esq., M.A., LL.D., has given his consent to a demonstration in the Hunterian Library of these valuable and beautifully illustrated volumes, at the coming meeting.

He has obtained for me a photograph of the first and last pages of this old English edition, which I trust you will be able to print along with this letter.

Yours truly,

W. B. INGLIS POLLOCK.

21, WOODSIDE PLACE,
GLASGOW, C.3.
November 29, 1946.

THE "NORMAL" IN THE SYNOPTOPHORE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—The interesting article by Lewis in your issue of December (*Brit. Jl. Ophthal.*, Vol. XXX, p. 749) reveals some results which are not in accord with my own experience.

Comparing the distribution of Maddox rod measurements with that in a series (which I hope to publish soon) of 1269 cases, certain features are apparent. Lewis' distribution is appreciably cramped at the greater levels of exophoria and esophoria. The mean is slightly more esophoric than in my series ($+0.895$ compared with $+0.508$). Using the Chi-squared test to compare distributions, the chance of obtaining Lewis' distribution from a population represented by my own series is less than one in ten thousand. This suggests that the Maddox rod measurements in Lewis' paper are not sufficiently reliable for conclusions to be drawn as to the relation between the Maddox rod and the synoptophore findings.

I venture to suggest that the failure to check the zero on the synoptophore is a matter of some significance in view of the conclusions drawn, since I do not think that there is, on the face of it, reason to suppose that the synoptophore and the Maddox rod measure different qualities of heterophoria.

I would also offer the following suggestion for the discrepancy between the findings of method A and method B with the synoptophore (and therefore for the bifurcation of the hump in the distribution curve:—

Intelligent anticipation would inevitably result in coincidence of the images being signified before true coincidence had taken place. With the slides used such anticipation could well be subconscious.

It is, I think, a fairly well established rule that the more simple the instrument, the less extraneous factors are introduced into a measurement, and thus the more likely is it that the measurement is truly that of the function being measured. The synoptophore is an instrument of recent development in comparison with the Maddox rod, and I have shown earlier (Cridland, 1940) that results with the Maddox rod can be made reliable by attention to details.

Lewis' investigation is a valuable contribution to synoptophore work, and my criticism seeks only to prevent too great reliance being placed upon the conclusions as to correlation between different instruments.

Yours faithfully,

NIGEL CRIDLAND.

SOUTHSEA, HANTS.

January 7, 1947.

MYOPIA AND PSEUDO-MYOPIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In the article "Myopia and Pseudo-Myopia" by J. P. Spencer Walker which appeared in the December issue of the Journal he mentioned the case of a boy, aged $13\frac{1}{2}$ years, who had

been rejected for Dartmouth owing to "myopia and myopic astigmatism." He found that the refraction under atropine was R. and L. $+0.25$ D.Sph. \bar{c} $+0.25$ D.cyl. vertical. He does not state whether or not as a result of his findings the boy was subsequently accepted for Dartmouth.

It would appear to me that it would not be in the boy's interest to be accepted, because it is probable that when he reaches the age to graduate from the college he would have become myopic, and might, therefore, be rejected at this late stage of his career. I would refer Mr. Spencer Walker, and anybody else who is interested in the subject, to an article in the American Archives of Ophthalmology of April, 1941, on "The Development and Prevention of Myopia at the United States Naval Academy" by Reginald Hayden, M.D. His researches would indicate that it is unwise for any boy to enter a naval academy who has not got at least a reserve of 0.5 D. of hypermetropia when refracted under a cycloplegic, and that visually such a candidate stands no better than an even chance of obtaining a commission in the line of the navy on graduation.

Yours faithfully,

F. S. LAVERY.

45, FITZWILLIAM SQUARE,
DUBLIN.

December 18, 1946.

OBITUARY

SIR RICHARD CRUISE, G.C.V.O.

SIR RICHARD CRUISE, G.C.V.O., joined the Staff of the Royal Westminster Ophthalmic Hospital, in 1909, the Committee of Management in 1923 and was appointed Consulting Surgeon in 1936. From 1918 to 1936, he was Surgeon in Ordinary to their Majesties King George V and Queen Mary and remained Queen Mary's oculist to the time of his death.

In 1934, he founded the Cruise Open Scholarship, of one hundred pounds per year, with a capital of £2000, which he had given and collected from his patients, to encourage research in Ophthalmology, at the Royal Westminster Ophthalmic Hospital. So far there have been three holders. Papers on the work done have been published in the *British Journal of Ophthalmology* from time to time.

Sir Richard was a first class operator especially interested in cataract extraction and glaucoma. For the former, he used the simplest possible technique but every detail even of "stance" was considered and perfected over years of practice. He used to teach

that one must "address" the eye as one would a golf ball. He strove to obtain the co-operation of his patient, saying that he was to consider himself, not the victim of a mutilating operation but the first assistant in a very delicate piece of craftsmanship. His results were almost invariably perfect.

In cases of glaucoma his work with the Bishop Harman scotometer and Schiötz tonometer, was a revelation to those unaccustomed to their repeated use.

He was for many years dissatisfied with the results of operative treatment for this disease and tried many techniques to obtain permanent drainage. At nearly the end of his life he was able to obtain an eye from a patient who had died an hour or two previously and prove that the drainage channels which he had produced by operation many years ago, had become lined by endothelium and so remained permanently open.

Sir Richard was always thoughtful for and helpful to his juniors and was ready to spend time and tireless patience in teaching them his own meticulous care in operative technique and persuading them to repeat these details themselves, in practice.

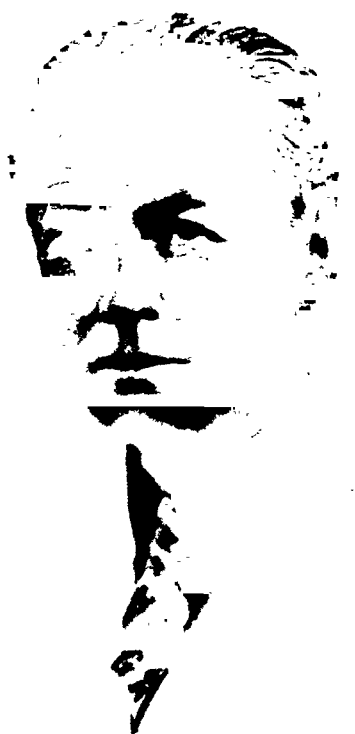
On the Committee of Management, he was a valued friend to the Medical Staff. While ready to champion any cause, he believed to be just, he had a clear idea of what could or could not be done. The excellent relations that maintained between the two Committees for many years were largely due to his tact and judgment.

He derived great pleasure from an outdoor life and regarded it as necessary to keep absolutely fit, if he was to do his best work as a surgeon. He was an excellent shot, fine cricketer, good golfer and rode well to hounds on his horse "War Gratuity." With him he won many hunt point-to-points.

During the twenty five years I worked with him, I came to rely upon his opinion as to what it was best to do in the "difficult" case with which one is faced from time to time, and he never failed me.

SIR WILLIAM J. COLLINS, K.C.V.O.

SIR WILLIAM JOB COLLINS died at his home in London on December 12, 1946, at the great age of 87 years. He was the eldest son of Dr. W. J. Collins, a London doctor, and the elder brother of the late E. Treacher Collins. Both brothers were educated at University College School and had among other contemporaries Mr. Percy Flemming and Ernest Clarke. Sir William's academic career was a distinguished one, both at School and at the University of London. He went to St. Bartholomew's Hospital for his medical training and graduated M.D. London in 1882, M.S. in 1885 and took the F.R.C.S.Eng. a year previously. He maintained his interest in the University all his life and was at times a member of the Senate and Vice-Chancellor.



SIR RICHARD CRUISE.

Sir William Collins was ophthalmic surgeon to the Temperance Hospital and at one time Surgeon to the Royal Eye Hospital. He joined the Ophthalmological Society of the United Kingdom in 1886, but never held office and retired some years ago. His outside interests were so many and varied that he did not often attend the Society's meetings. In this there was a marked contrast between the brothers, for Treacher Collins hardly ever missed a meeting and apart from his work had few outside interests. Thus, it is not as an ophthalmologist that Sir William will be remembered, but for his work on the L.C.C. and as member of Parliament. How few to-day remember that London largely owes its ambulance service to his untiring efforts. District Nursing was another concern in which he took great interest and the Chadwick Trust.

NOTES

Death As we go to press we learn with great regret of the death of Sir Arnold Lawson. We hope to publish a memoir in a later number.

* * * *

Honours In the New Year's Honours List we are pleased to see that the C.V.O. has been conferred on Mr. F. A. Juler.

MR. EUGENE WOLFF has been elected an honorary member of the Société belge d'Ophthalmologie.

* * * *

Appointment MR. J. D. MARTIN-JONES has been appointed Ophthalmic Surgeon to the Salisbury General Infirmary, Wilts.

* * * *

The British Orthoptic Journal THE third number of this journal is just as interesting as its predecessors, if not more so, and it possesses the same freshness of outlook. Those who are inclined to scoff at "exercises" might be interested to read Miss Sparrow's paper on the "Art of Seeing," and Miss Mayou's on "Convergence Deficiency," while a dilemma, which was frankly inexplicable by the authors of an article in a recent number of the *American Archives*, is neatly resolved by Miss Swift and Miss Balkwell. It concerns the apparent difference in distance of red and green lights, which may often be noted in, say, the "Friend" test.

That orthoptics is coming into its own would seem to be shown by the provocative statement of one ophthalmic surgeon, that "the surgeon must work for the orthoptist—not the orthoptist for the surgeon."

The remainder of the articles are not of this character, however, though some of them make a justifiable plea for early operation in cases of squint.

It is impossible, within the limits of a short review, to deal with each of the seventeen papers by ophthalmic surgeons and orthoptists, but we cannot conclude without mentioning "Doubts and Difficulties" by Miss Exner, which, from a delightfully modest beginning—"I very nearly didn't come,"—goes on to give much useful information, and is full of sound common sense.

The circulation of this journal is at present small, but the material in it is of such value, that we feel the venture of publishing it should be encouraged, and that it would not be out of place to state that copies, 5/6 each, including postage, may be obtained from the Publishers, Wilding and Son, Ltd., Castle Street, Shrewsbury.

* * * *

National Society for the
Prevention of Blindness.
Prize Subject: Glaucoma

THE National Society for the Prevention of Blindness announces that papers submitted for the glaucoma prize of \$500 offered in 1944 did not conform to the criteria set up by the ophthalmological committee selected to award the prize. Therefore, the prize is again offered for the most valuable original paper adding to existing knowledge about the diagnosis of early glaucoma or the medical treatment of non-congestive glaucoma. The criteria may be secured by writing to the National Society for the Prevention of Blindness, 1790 Broadway, New York 19, N.Y.

Papers may be presented by any practicing ophthalmologist of the Western Hemisphere and may be written in English, French, German, Italian, Spanish or Portuguese. Those written in any of the last four languages should be accompanied by a summary in English. Closing date for receipt of papers is December, 1947.

The award will be made by the Society with the guidance of an ophthalmological committee composed of Doctors John N. Evans, *Chairman*; Frank C. Keil, Daniel B. Kirby, John M. McLean, R. Townley Paton, Algernon B. Reese, Bernard Samuels, Kaufman Schlivek, Willis S. Knighton, Manuel Uribe Troncoso, David H. Webster.

* * * *

Western Ophthalmic
Hospital

MR. LINDSAY-REA retired from his post on the staff of the Hospital on December 13, 1946, after 25 years service. His colleagues made a presentation to him on his retirement.

THE BRITISH JOURNAL OF OPHTHALMOLOGY



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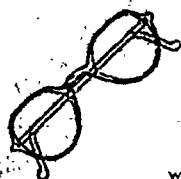
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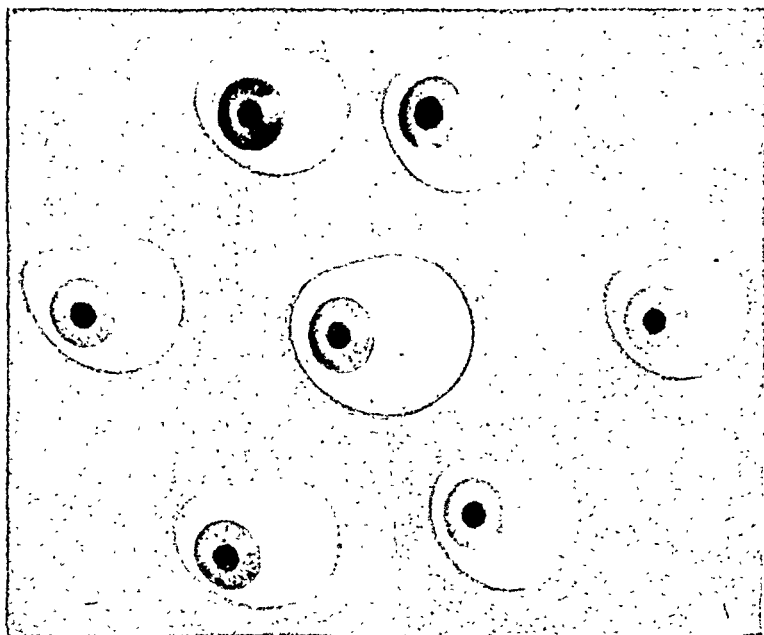
THE BRITISH JOURNAL OF OPHTHALMOLOGY LTD.

24-27, THAYER STREET, W.1



ARTIFICIAL EYES IN PLASTIC

Artificial Eyes until the beginning of the war were invariably made of glass, and their production was entirely in the hands of a few very skilled craftsmen in this Country and on the Continent. Up to August, 1939, Theodore Hamblin, Ltd., employed Mr. Paul Asprien, of Vienna, at 15, Wigmore Street, and at their various provincial branches, where he made artificial eyes in glass while the patient waited. With the outbreak of war, these visits had to cease and steps were taken to develop the manufacture of artificial eyes in plastic material. The many difficulties of producing eyes in this material have been overcome, and they are now made throughout in plastic, no paper or glass being incorporated.



Eyes made in plastic have many advantages over those made in glass. They are life-like in appearance, comfortable in wear, are not affected by the secretions of the orbit, and above all, they are unbreakable.

Difficult shapes necessitated by war injuries, burns, etc., or thin shells to fit over shrunken or deformed globes, almost impossible to produce in glass, are quite possible in plastic.

Patients may be sent in to 15, Wigmore Street or to most of the provincial branches, where stocks of ready-made eyes are available from which selections may be made and fitted. Specially made eyes for more difficult orbits can be made with little delay. For these a carefully made mould of the orbit is first taken, and a special iris is produced in plastic. In such cases a second visit after the eye has been made is necessary for fitting.

**THEODORE
HAMBLIN LTD**
DISPENSING OPTICIANS
15 WIGMORE STREET,
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THE BRITISH JOURNAL OF OPHTHALMOLOGY

MARCH, 1947

COMMUNICATIONS

THE COMPARATIVE VISUAL ACUITY AND EASE OF READING IN WHITE AND COLOURED LIGHT *

BY

L. C. MARTIN AND R. W. B. PEARSE

THE subject of visual acuity continues to attract attention. The experimental facts of contour acuity and the certainty of the relatively diffuse character of the light concentration in the retinal image in comparison with the size of the retinal elements, together with the supposed rapid perturbations of the eye direction, have made it reasonable to conclude that visual acuity is dependent on local brightness discrimination, which is known to be a function of the intensity level. We need not here discuss the operation of the retinal mosaic except in so far as it would seem to set a lower limit to resolution under certain special conditions.

The chromatic aberration of the optical system of the eye will render a "white" image somewhat more diffuse than the monochromatic image, and we may reasonably anticipate some improvement of acuity, "other things being equal," in using monochromatic light. Certain measurements of relative visual acuity in monochromatic v. white light have, however, yielded contradictory results. The experiments now to be described were not conducted primarily with a view to testing this point, but for a more technical purpose.

* Received for publication, January 10, 1947.

Nevertheless, they may be found of interest in their bearing on the general problem. It was desired to obtain information on the *relative* acuity of an accommodated eye both in red and white light, and also the comparative "ease of reading"; the former presents the more definite problem. The conditions imposed on the work precluded the use of artificial pupils, and binocular vision was used throughout. It was desired, however, to pay careful attention to

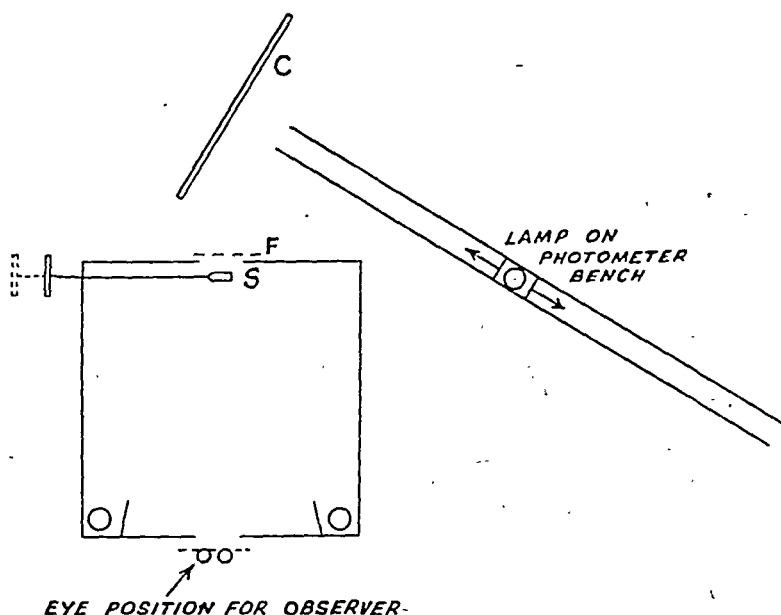


FIG. 1.

the level of brightness of the visual field and the consequent state of adaption of the eye.

The apparatus designed for this purpose, Fig. 1, consisted of a cubical white-lined rectangular box, each edge of which was about 20 in. long. One wall is pierced by the observing aperture (about 3 in. by 1 in.), and the opposite one by a square hole, subtending about 8.5° in the observer's visual field, through which the background C of the acuity object was viewed. The background field was obtained by illuminating a white card by suitable lamps (of colour temperature approximately 2680°K) moving on a long optical bench. The illumination of the white field was measured by a Holophane lumeter. A wide range of illumination levels could be obtained. Red or neutral filters could be placed over the hole at F so as to modify the illumination of the central field. The spectrophotometric transmissions of these filters (Fig. 2) were

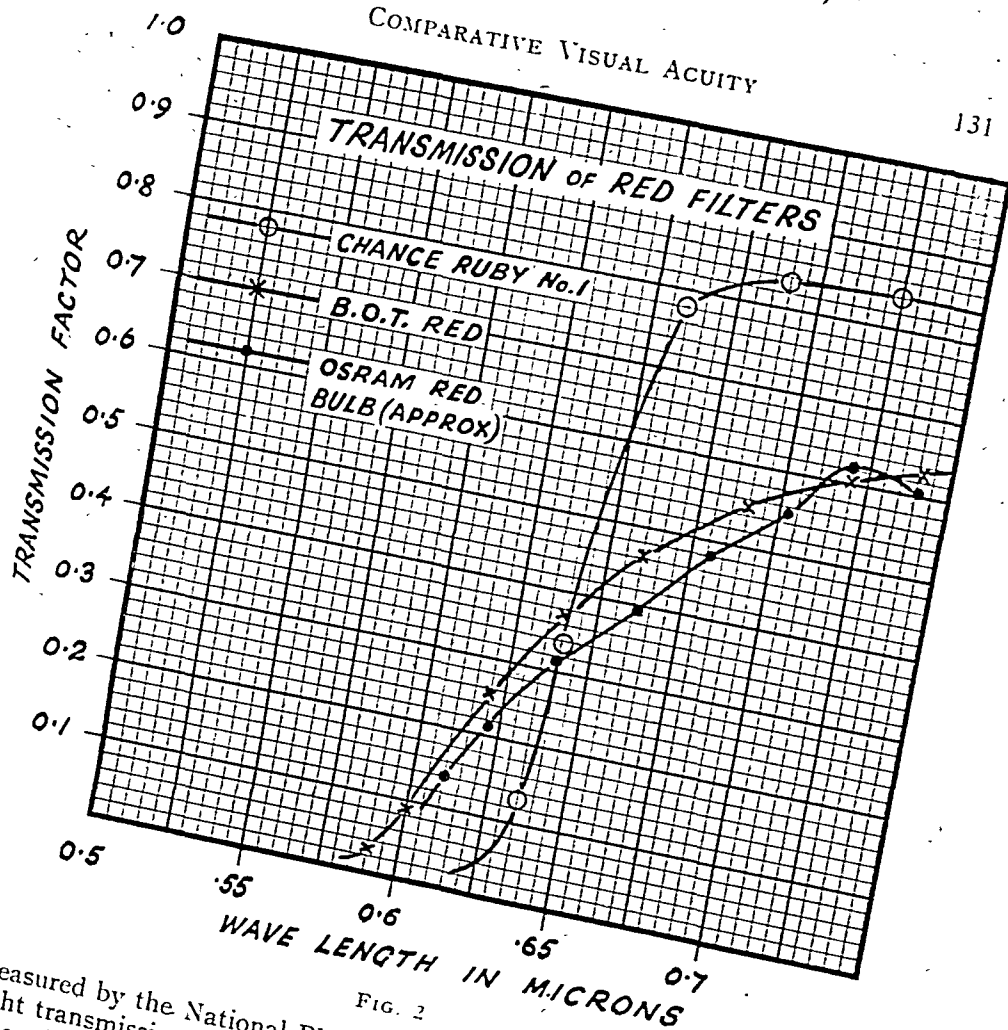


FIG. 2

measured by the National Physical Laboratory, and their *apparent* light transmission computed with standard photopic visibility data. The white card is assumed to reflect non-selectively.

The specification of the illumination of the field then avoids heterochromatic photometry.

The box contained both red and white lamps screened from direct view, but capable of illuminating the matt-white interior wall of the box (subtending about 30° each way) in which the observing hole is pierced. The illumination level of this wall is thus controlled by screens and rheostats so as to be as nearly equal as possible to and of the same general colour as that of the central field for all observations. The forehead of the observer rested against a velvet-covered stop so that the distance of observation was kept constant. The acuity object (Fig. 1), was generally a grating; a small transparency about 1 cm. square cut from a lantern slide on which

a half-tone screen (55 lines per inch.) had been printed. There are well-known disadvantages in the use of a grating object with many lines, always parallel to one direction, in any attempt to obtain a fair estimate for the fundamental limiting angle of visual acuity. On the other hand, since the primary object was the *comparison* of the red and white illumination conditions, the great convenience of a grating object seemed to outweigh its limitations. The possibility that a grating may give anomalous results near the threshold by appearing with reversed contrast was not overlooked, and a good many trials were made with a much smaller object of the Cobb type, which seemed, however, to offer undue difficulty to some observers. No evidence that the grating was giving anomalous results came to light.

This object was only adopted after trials with other systems, in some of which reduced images of other acuity objects were projected into the binocular visual field by mirrors or lenses.

TABLE I
Visual Resolving Limit in Minutes of Arc
("B.O.T. Red" and "White")

Observer	Field Brightness Level (Foot-candles)						
	2.64 Red	3.30 White	0.245 Red	0.295 White	Observer*	0.050 Red	0.060 White
2	1.64	1.69	1.94	1.92			
5	1.47	1.57	1.75	1.92			
11	1.48	1.52	1.93	2.14	11	2.32	2.75
13	1.80	1.93	1.89	1.97	15	2.29	2.39
12	1.85	1.96	2.05	2.00	12	2.19	2.15
10	1.65	1.64	1.90	2.00	10	2.27	2.32
3	1.81	1.72	2.10	2.33	3	2.40	2.65
1	2.20	2.20	2.27	2.26	21	2.66	2.73
8	1.90	1.88	2.06	2.08	22	2.77	2.96
9	1.55	1.57	1.71	2.18	20	2.07	2.23
6	2.19	2.22	2.66	2.68	6	2.55	2.60
4	1.75	1.76	1.89	2.21	19	2.46	2.69
Average ...	1.77	1.81	2.01	2.14		2.4	2.55
Relative Acuity	0.564	0.552	0.498	0.467		0.417	0.392

*The results for the lowest brightness of field with the B.O.T. Red are not strictly comparable with the others, since some of the observers differ. The programme at first included only two brightness levels, and some who took part in the first observations were not available later.

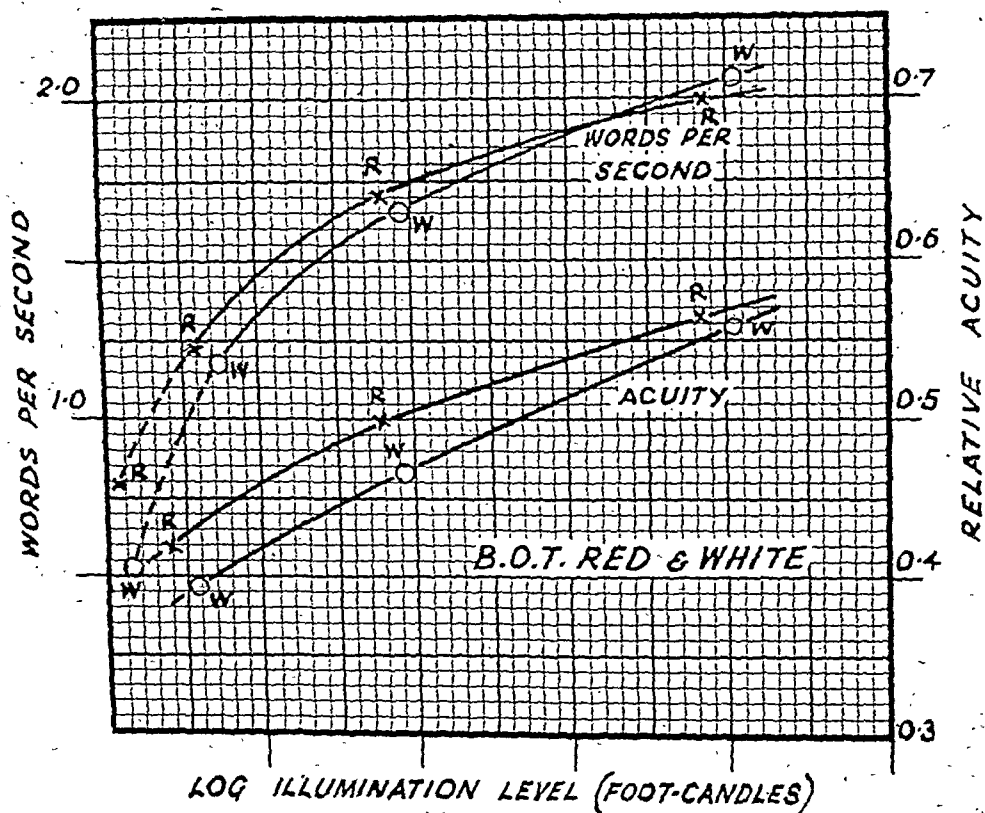


FIG. 3.

Ease of reading and acuity; B.O.T. Red and White.

Comparisons were made between (a) white light (b) light transmitted by Chance's Ruby No. 1 glass, and (c) light transmitted by Board of Trade Red glass. The general levels of illumination used were approximately: 0.06, 0.6, 6.0 foot-candles (and for some experiments 3.0 f.c.). Exact values appear in the tables.

The red and white observations were sandwiched, four or five trials being made with white, then with red, then with white again; all for one level of illumination. The illumination was then changed and the procedure repeated.

In the first set of trials the recorder tilted the grating in steps of 10° till the observer reported resolution ceased; he then went back 20° and advanced in steps of 3° ; then back 10° and advanced in steps of 2° till the apparent limit was ascertained. However, this system seemed to throw a good deal of strain on the observer, and it was abandoned in favour of finding quickly a first rough setting, and then changing the setting by a degree or two as might seem necessary till the observer feels that the limit is reached. The

observer is allowed to sit back from the observing position for a few minutes as often as he feels it necessary to avoid any feeling of strain in position or attention. The observer did not himself adjust the setting of the grating. He had only to report whether he could resolve it or not. The observers, who varied in age from 14 to 50 years, wore their normal spectacles, if any.

The results with "B.O.T. Red" and "White" are given in Table I and Fig. 3.

The measure of acuity is plotted as the reciprocal of the visual resolving limit in minutes; this limit is the angular subtense in the observer's visual field of the apparent size of one grating element, i.e., the apparent angle between the mid-points of two adjacent bars or spaces, taking account of the tilt.

The results with Chance's 'No. 1. Ruby' and 'White' are given in Table II and Fig. 4.

TABLE II
Visual Resolving Limit in Minutes of Arc
(Chance's "Ruby, No. 1" and "White")

Observer	Field Brightness Level (Foot-candles)					
	6.3 Red	6.3 White	0.66 Red	0.63 White	0.066 Red	0.063 White
8	1.17	1.17	1.35	1.51	2.21	2.31
15	1.60	1.63	1.71	1.98	1.98	2.74
3	1.38	1.46	1.45	1.53	1.98	2.31
10	1.24	1.24	1.36	1.41	1.61	1.82
12	1.07	1.09	1.19	1.16	1.54	1.53
11	1.24	1.20	1.34	1.54	2.03	2.46
24	1.61	1.63	1.68	1.76	2.02	2.32
23	1.98	1.91	1.83	1.77	2.55	2.70
19	1.17	1.24	1.55	1.56	1.79	2.18
26	1.67	1.71	2.30	2.32	2.07	2.41
25	1.52	1.53	1.70	1.74	2.39	2.54
Average ...	1.42	1.43	1.59	1.66	2.01	2.30
Relative Acuity	0.70+	0.699	0.629	0.602	0.498	0.435

It was unfortunately not possible to ensure that identically the same group of observers was used for the acuity observations with the two red glasses, moreover the apparent general improvement in the results with the Chance's Ruby glass probably indicates some effects of practice. In spite of the discrepancies between the general averages for the two sets of observations it is probable that the red and white observations have been affected equally.

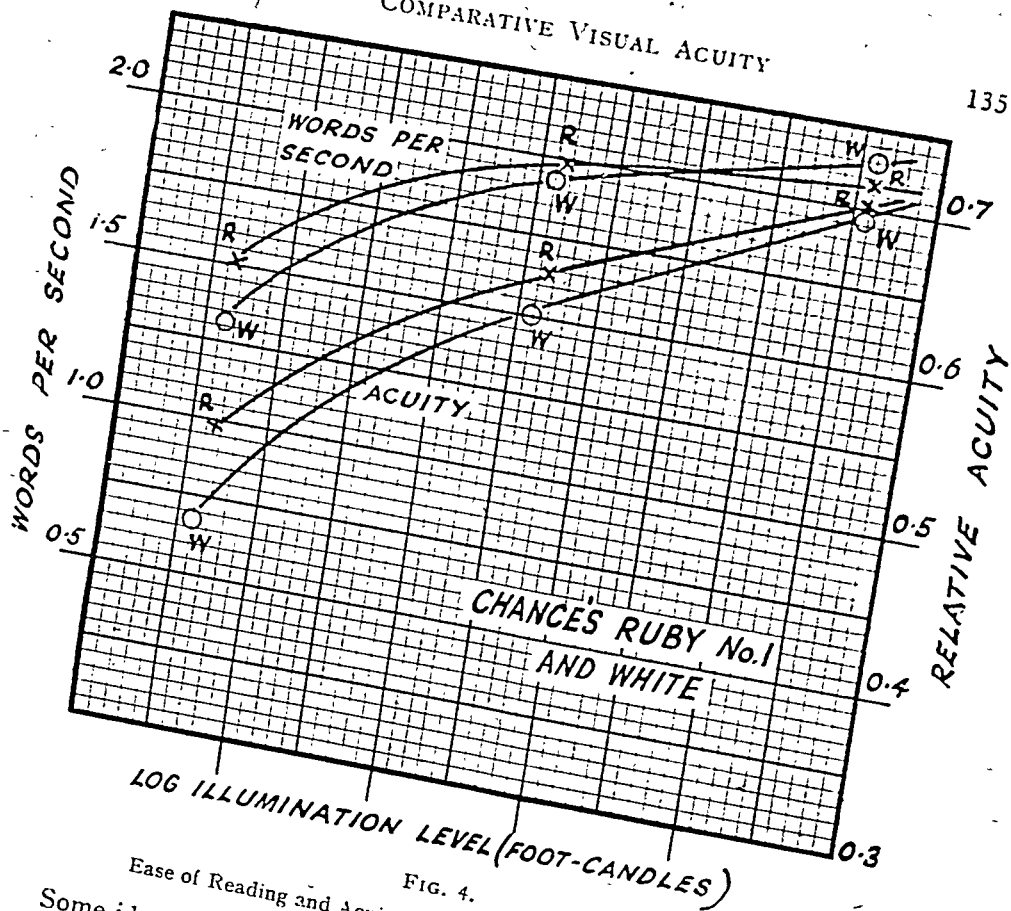


FIG. 4.
Ease of Reading and Acuity; Chance's Ruby No. 1 or White

Some idea of the consistency of the readings (in the second series above) can be obtained from the following table, which shows the spread of the angular settings for three observers picked at random. The columns correspond to the table above.

TABLE III
Difference between Highest and Lowest Angular Settings in Seven Readings together with the Mean Reading

Observer	6.0		0.6		0.06	
	Red	White	Red	White	Red	White
25						
26	3/61°	5/61°	5/57°	2/56°	7/40°	8/36°
19	3/58	1/59	7/43	2/60	12/48	4/40
	3/68	3/67	2/66	2/60	3/46	2/55

As in the case of the reading trials it would be meaningless to make a formal statistical analysis of the results, since it had been made clear that the immediate result is a function of the experience and visual condition of the observer. But to give one example, and having regard only to the variation of the results, the "probable error" of an estimated angular setting for a typical observer (W.D.W.) is only about 0.5° when the angle is 46° ; this in turn means about 0.02 minutes in the resolving limit, or about one per cent. This accuracy should be carried over at least into the comparison of red and white illuminations.

For all these figures and curves it may be concluded that

(a) the angular resolving limit is about 4 per cent. better for the average observer at 0.6 ft/candles for red than for white of equal illumination;

(b) for most (but not all) observers the advantage of the red light is more marked at still lower levels of illumination.

(c) For equal visual acuity about 80 per cent. more white light than red is required in the range 0.3 to 0.5 f.c. and at lower levels the ratio of white to red (for equal acuity) may exceed 2:1.

(d) There is no significant difference between the two kinds of red light.

The general results thus indicate that while there is some suggested improvement in using red light rather than white at low illumination levels, the answer to the question as to the influence of the chromatic aberration of the eye on acuity cannot be answered with any certainty owing to the marked influence of intensity on acuity and the uncertainties of heterochromatic photometry.

The visual acuity of the unaccommodated eye for different colours under conditions of high brightness

The result that intensity rather than colour was the major controlling factor in acuity in the laboratory experiments suggested that acuity of vision when using colour filters in conditions of high illumination might be similarly affected.

Following the experience of the laboratory test, a rotating grating acuity test object was used. It had horizontal lines, and rotated about a horizontal axis. The grating had an element size 16 mm., and was mounted between two opposite apertures in a box through which the grating could be seen against a background of sky. The observer's distance varied from 100 ft. to 140 ft. according to conditions. The front of the box was hidden by a mirror having an aperture 5.8 cm. high and 7.0 cm. wide corresponding to that in the box behind it, and the mirror was adjustable so that it reflected an image of the sky near the horizon behind the observer. In this way

the immediate surrounding field for the acuity object could be given the same general brightness as the sky background. The recorder 'set' the grating, and the observer signalled his observation.

The following filters and goggles were employed:—

Filters			Percentage Photopic transmission (daylight)
1.	Kodak hand-shade Yellow	67
2.	Kodak hand-shade Orange	30
3.	Red eye-shade	16
4.	Ilford minus Green	13
5.	Neutral goggles	5
6.	Kodak hand-shade neutral	3
7.	Goggles with plain glass	90
8.	Ilford Yellow-green (7)	45
9.	Ilford Blue (804)	40
10.	Special double filter (purple) (used as hand-shade	2.2
11.	Olive green (946) hand-shade	77

All the filters, except where hand-shade is specified, were used in goggles.

Observations for the high and medium brightness levels were made on the roof of the Imperial College with a general direction of view East to West, and mostly between 10.30 a.m. and 3.30 p.m. (Summer time) so that the sun is to the left. Observers were allowed to shade their eyes from direct sunlight. The 'medium' level observations were made on very dull days.

The brightness of the sky near the acuity object was observed with a Holophane lumeter, but the instrument was working near the top of its range and the readings are approximate only. The readings in apparent foot-candles are given in the following tables. Observations for the lowest brightness were made *indoors* in a corridor; the grating was seen against a white screen with daylight illumination, and a matt white surround field was employed.

Observations were made for each filter and each brightness level as follows:—

1. Three settings with naked eyes.
2. Three settings with filter.
3. Three settings with naked eyes.
4. Three settings with filter.
5. Three settings with naked eyes.

Thus each figure given in the table for a result "with filter" is the mean of six settings sandwiched between nine settings with naked eyes; the mean of these nine is also entered.

	Neutral Goggles D = 1.3		K.H. S. Neutral D = 1.5		Plain Glass Goggles		Ilford Yellow Green (Y)		Ilford Blue (804)		Double Filter Purple		Olive Green Hand- Shade	
	with	with- out	with	with- out	with	with- out	with	with- out	with	with- out	with	with- out	with	with- out
1. P.	1'36	1'08	0'97	0'86	1'44	1'43	1'04	1'05	1'02	1'04	1'41	0'85	1'28	1'25
													0'72	0'772
2. M.	1'63	1'24	1'23	0'87	1'18	1'28	1'38	1'38	1'35	1'35	1'99	1'38	1'29	1'24
													1'27	1'32
3. B.	—	—	—	—	0'48	0'58	0'96	1'01	1'14	1'14	—	—	—	—
4. S.	—	—	1'50	1'44	—	—	—	—	1'32	1'31	—	—	—	—
5. M.	1'87	1'27	—	—	—	—	—	—	—	—	—	—	1'20	1'15
6. M.	—	—	—	—	—	—	—	—	—	—	3'00	1'98	2'38	2'22
7. M.	—	—	—	—	—	—	—	—	—	—	—	—	1'20	1'24
	1'62	1'20	1'23	1'06	1'03	1'10	1'13	1'13	1'21	1'21	2'13	1'40	1'33	1'31

Brightness levels of Observations above in f. c.

1.	1,000	2,500	1,200	2,000	2,000	1,000	400 1,000
2.	1,000	2,500	2,200	1,000	2,000	1,000	400 1,000
3.	—	—	2,500	2,000	2,000	—	—
4.	—	2,200	—	—	2,000	—	—
5.	800	—	—	—	—	—	800
6.	—	—	—	—	—	1,50	1,500
7.	—	—	—	—	—	—	2,000

The most prominent feature of the observations is the loss of acuity with the majority of the filters; the loss is generally greater, the greater the absorption. Plotting the mean results of R.W.B.P. and L.C.M. for naked eye vision against log. of field brightness a curve was obtained from which the expected increase of angular resolving limit for any filter consequent on its known diminution of brightness can be roughly found. The data are:—

Illumination in equiv. f. c.	Angular Resolving limit
1,700	0.96
75	1.30
1.0	1.64

Other published data (Lythgoe, Hecht and others) show considerable mutual divergences, and obviously depend greatly on experimental conditions. Our experimental results are therefore retained as likely to apply (better than others) to the conditions special to our work. The following table shows the expected ratio of resolving limits thus anticipated from the loss of light, compared with the observed values; they relate to the highest brightness level only.

Ratio of Resolving Limits

Filter	Transmission factor	Calculated from experi- mental results	Observed
K.H.S. Orange ...	0.67	1.05	1.07
K.H.S. Yellow ...	0.30	1.14	1.10
Red eye-shade ...	0.16	1.21	1.09
Ilford Minus green ...	0.13	1.23	1.08
K.H.S. Neutral ...	0.03	1.36	1.16
Neutral goggles ...	0.05	1.32	1.36
Ilford Yellow green ...	0.45	1.10	1.00
Ilford Blue (80+) ...	0.40	1.12	1.00
Double filter (purple)...	0.025	1.37	1.52
Olive green (AP 946)	0.77	1.04	1.02

The adoption of Lythgoe's results for the variation of acuity with brightness would lead to somewhat smaller 'expected' values; in

fact the 'expected' values for the red eye-shade would fall to 1.13. It may be concluded that as far as the present experimental evidence goes, the whole 'acuity' deterioration caused by a filter may be attributed to the loss of light. This 'photometric' sensitiveness makes outdoor observations very difficult to conduct unless conditions are very steady. Adaptation is very noticeable in the first few moments after putting up goggles or hand-shade, but it cannot be measured with any certainty under the conditions described above.

It may be of interest to note that the observer L.B., who reports extraordinarily small resolving limits as compared with other observers has shown himself to be an exceptionally good rifle shot. In spite of all that has been done on 'acuity', this work suggests that there are still questions regarding effects in daylight (and for particular observers) which are not well understood. The results of L.B. were very consistent, and his estimates were tested over and over again under conditions in which it was impossible for him to know what results his answers would indicate.

Exploratory tests on "Ease of Reading" in Red and White illumination

The general subject of "ease of reading" is one which has received considerable attention from psycho-physical experimenters in connection with investigation of the optimum illumination, size and form of type, and other factors. Apart from the investigation of the possible speed of reading under various circumstances, attempts have been made to register the necessary effort put-forward by the reader in terms of "rate of blinking," "muscular tension," "eye movements and the indicated span," "changes in pulse rate" and so on, but it appeared from a first survey of the literature that it would be dangerous to base any experimental conclusion on criteria of the above kind unless as the results of very protracted work. In this case the objective was quickly to obtain a comparison of the ease of reading in equal red and white illumination, and it was decided after consultation with workers who are familiar with such investigations to compare the numbers of simple disconnected words readable in short-period exposures, alternately red and white. Careful consideration was given to the provision of the alternating illumination, and the avoidance of any extraneous factors (such as glare from a page of paper, etc.) which might, if undetected, produce an erroneous result.

The reading material consisted of plainly printed simple words arranged in 15 groups and photographed as lantern-slide transparencies. The slide could be placed in a holder over the viewing hole at S, Fig. 1. The order for exposure of five or seven of the groups was selected arbitrarily before any seven tests; the colour was changed between each group.

The observer, looking through the observing aperture, saw the surround field and fixated a small mark about 10° below the lower edge of the reading field; the latter was quite dark until a switch actuated by an 'Advertisement' motor was mechanically closed for a fixed time of about seven seconds.

The observers were asked to read at such a speed that they felt reasonable accuracy would be possible; occasional casual errors were not heeded in recording the number of words read. The selection and order of the groups, and the order of the relative illumination levels differed from person to person. These precautions, together with the sandwiching of red with white observations, should result in the ultimate elimination of the effects due to 'experience' and the inevitable effects of variations in the reading material.

There is a noticeable lag in the starting of reading after the appearance of the light. Once the reading starts, the observer does not feel much difference of effort between, say six foot-candles and 0.6 foot-candles, but it is considered that the eye movements involved in normal reading, in going from the end of one line to another, are likely to be hindered by loss of illumination; this may be compared with the difficulty in commencement of reading when the light is switched on. The initial lag may therefore fairly be regarded as a part of the test and not merely as an error.

The results for several observers using the B.O.T. Red alternately with white, and Chance's Ruby No. 1. alternately with white are given in the following tables; see also Figs. 3 and 4.

TABLE V
Average Number of Words per Second "Red (B.O.T.)" and "White."

Observer	Level of illumination (Foot-candles)							
	2.64 Red	3.30 White	0.245 Red	0.295 White	0.060 Red	0.072 White	0.034 Red	0.038 White
6	1.85	1.82	1.85	1.88	1.16	1.12	0.78	0.51
4	1.88	1.92	1.92	1.96	1.51	1.51	—	—
2	1.88	2.03	1.72	1.47	1.02	0.86	—	—
5	2.33	2.19	1.65	1.51	—	—	—	—
11	1.96	2.13	2.12	1.78	1.19	1.30	—	—
13	2.23	2.13	1.85	1.92	—	—	—	—
12	2.47	2.47	1.69	1.92	1.48	1.33	—	—
10	2.33	2.19	1.69	1.58	1.37	1.37	—	—
15	1.33	1.72	1.23	1.19	—	—	—	—
8	1.78	1.85	1.36	1.44	0.71	0.59	—	—
9	1.96	2.22	1.58	1.55	1.37	1.37	—	—
Mean for all observations	2.00	2.05	1.70	1.65	1.23	1.18	0.78	0.51

TABLE VI.

Average Number of Words per second "Chance's Ruby, No. 1 and "White"

Observer	Levels of illumination (Foot-candles)					
	6'3 Red	6'3 White	0'66 Red	0'63 White	0'066 Red	0'066 White
8	1'92	1'88	2'20	2'10	1'0	1'09
15	2'22	2'33	2'10	1'85	1'58	1'37
10	2'10	2'13	2'10	2'12	1'19	1'16
12	2'58	3'19	2'30	2'20	1'72	1'47
11	2'24	2'33	2'01	2'22	1'41	1'12
24	1'33	1'34	1'58	1'51	1'40	1'27
23	1'82	1'71	1'74	1'72	1'74	1'64
19	2'22	2'33	1'99	2'01	1'54	1'37
26	2'15	2'19	1'82	1'78	2'03	1'78
25	2'19	2'05	2'01	1'18	1'40	0'82
Mean	2'08	2'15	1'98	1'93	1'50	1'31

In estimating the precision of the results, typical values as obtained by one observer (J.P.) may be quoted. Results are the actual numbers of words 'read' in the trials; all of the same duration.

Approximate illumination level, foot-candles					
White	0'06	Red	White	0'6	Red
7			17		17
		12		15	17
11		9	15		16
8		10	15	18	16
7			18	17	
Mean	8'2	10'3	16'2	14'7	17'0
					16'3

The number of results is insufficient for any satisfactory statistical analysis, and indeed the numerical results for speed of reading represent a quantity which depends on very complete psychological factors. However, the quick alternation between white and red was

an effort to secure that whatever the uncertainty in other things, any difference between these illuminations should be brought out. The numerical results suggest that his reading speed is being measured by each observer under any one set of conditions with an error which does not often exceed one word in 7.3 seconds or 0.14 words per second. If there be an average reading speed for human beings it certainly cannot be found from experiments from ten to twelve observers; on the other hand if there be any consistent difference for red and white it should remain (like the effect of varying brightness) after averaging for such a number. It may be concluded that ease of reading and relative acuity show similar variations under the conditions of these experiments. In particular:—

(a) Any difference between red and white illumination in their effect on ease or speed of reading is too small to be certainly established in the present experiments at levels of 0.3 or 6.0 foot candles, though there is some indication that "white" may have some advantage at the higher and "red" at the lower of these two levels.

(b) The speed of reading at the lowest intensity (0.06 foot candles) is about 15 per cent. greater for red than for white at equal illumination levels. For equal reading speeds it is necessary to have about 60 per cent. more white than red light.

(c) There is no appreciable difference between the two types of red light at any level. The effects of variation with intensity agree fairly well with "speed of reading" data published by Luckiesh, Taylor and Sinden (*Jl. Franklin Inst.*, Vol. CXCII, p. 757, 1921).

As mentioned above, the purpose of these experiments was of a practical nature, and no attempt was made to use artificial pupils. The eye is under-corrected for both colour and spherical aberration, but the zone of maximum under correction is of radius 1.5 to 2.0 mm. Changing from white to red illumination will remove the chromatic aberration, and call for an increase of about 0.5 D in the effort of accommodation; this is likely to result in a small contraction of the pupil, which would improve the definition but lower the illumination as compared with any possible experiments using artificial pupils. It is not known whether there was any change in pupil size consequent on changing from red to white, or vice versa, in our experiments.

Only one case of marked colour defectiveness, No. 26, deuteranomalous, was known amongst our observers, though a formal test of colour vision could not be given to all. It is well known, however, that 'normal' individuals differ somewhat among themselves in the apparent luminosity assigned to deep red light ($\lambda > 0.65\mu$) as compared with white. If it had been possible to find the relative acuity

etc., with respect to the true apparent luminosity in all cases, the results might have been considerably modified. It is possible that the small relative advantage in acuity and ease of reading associated with red light can be attributed to the improved definition and contrast of the monochromatic image, but there does not appear to be any ready explanation of the tendency of this advantage to disappear, and even reverse, with increasing intensity. The Complex factors are at work which effectively prevent any hasty judgment as to the relative merits of red and white illuminations for acuity and ease of reading. Acuity is, however, not the only desideration for ease of reading, as soon appears from subjective texts.

Subjective tests of reading in red illumination

1. For the first part of these trials a number of red electric lamp (G.E.C. Osram ruby 40 watt) bulbs were procured and loaned to selected individuals with the request that each one should read for half an hour or more in the red light, and give a report of his experience. These bulbs are of ruby glass (not 'sprayed') and the light is largely confined to wave-lengths greater than 0.65μ .

In all cases a reading lamp was employed. An illumination of approximately 0.25 apparent foot-candles can be obtained at about 18 ins. from these lamps with the red bulbs, but they vary among themselves to a considerable extent, and in some cases the illumination did not exceed 0.1 apparent foot-candles.

As a general result of the tests there seems to be no real difficulty in reading at any illumination from 0.1 f.c. (apparent) upwards, except that one or two people find a slight difficulty in *beginning* to read. The 'surroundings' seem dark, and there is in some cases a rather vague unfavourable mental reaction—possibly associated with a slight degree of extra effort at the 0.1 f.c. level. Those who tried reading at higher illuminations did not mention any difficulty; it seemed considerably more attractive to read at 1.0 f.c. than at 0.1 f.c.

The para-fovea is well known to be less sensitive to red light as compared with the shorter wave-lengths, and the surroundings appear very feebly illuminated as compared with the book. This again makes conditions seem a little unfamiliar. It may be expected that red light will not be very satisfactory (from a "safety" point of view) in the neighbourhood of machinery, etc.

Some efforts were made in the subjective tests to determine how many words could be read in red light in 15 minutes as against white, but it was found that the uncertainties associated with the variation of the reading material, the changing adaptation of the observer, the effect of practice and so on, were so great as to mask any difference attributable to the change of colour of the light, and the attempt was abandoned in favour of the short-period exposure tests which have been described above.

ALLERGY TO ENDOGENOUS HORMONES AS A
CAUSE OF KERATITIS ROSACEA*†

BY

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ZONDEK and Bromberg¹ have found that patients suffering from typical allergic symptoms (asthma, rhinitis vasomotoria, urticaria, angioneurotic oedema, etc.) and certain other complaints not usually classified as such (premenstrual tension, pruritus vulvae, migraine, etc.) show in a certain percentage of cases hypersensitivity to their own hormones ("endocrine allergy"). In an earlier paper observations on women with symptoms depending on the genital cycle and related to menstruation or menopause were reported. It was suggested, however, that hypersensitivity to endogenous hormones can also occur in men and women quite independently of genital function.

The condition of endocrine allergy can be detected by an active intracutaneous test. The reaction to a steroid hormone is considered positive when 24-48 hours after intracutaneous injection of 0.1 mg. of steroid hormone dissolved in 0.1 c.c. of specially purified olive oil, a red or pink, slightly elevated and frequently itching papule occurs at the site of the injection. Reactions to insulin (0.1 c.cm.=0.01 units) or gonadotropin (0.1 c.cm.=0.1 units) are read as positive when 1-2 hours after injection of the hormone in physiological saline solution a wide erythema with urticarial swelling occurs at the site of the injection. The characteristic local reaction is accompanied in certain cases by a general reaction (angioneurotic oedema, urticaria, rhinitis vasomotoria, etc.) which affords further confirmation of the diagnosis.

Presence of specific allergy antibodies (reagins) in cases of endocrine allergy has also been demonstrated by the passive transfer test according to Prausnitz and Kuestner, and by spontaneous endogenous passive transfer tests.^{1,2}

The cases selected for the original series of examinations were all of unknown aetiology from a group which had been thoroughly investigated, and in which a condition refractory to treatment had persisted for a considerable period (more than 2 years). In several of the cases desensitisation treatment was applied. Good results were obtained by this means in a large percentage of the group. Details of the treatment and the technique applied have been presented in earlier papers.^{1,2}

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Certain eye diseases of still unknown aetiology (like a case of undetermined keratitis superficialis) were also investigated in the course of this study in endocrine allergy, and good therapeutic results were obtained by desensitisation treatment with allergenic hormones.^{1, 2}

The numerous theories of the pathogenesis of keratitis rosacea seem to point to the lack of any definite knowledge as to its true aetiology. It seemed to us that allergy to endogenous hormones might account for a number of these cases.

Keratitis rosacea is characterised by garland-like dilatation of the limbal vessel, vascular invasion of the cornea, and appearance of sub-epithelial opacities in the anterior corneal layers. The opacities gradually increase and finally are apt to cause impairment of vision. The subjective symptoms include photophobia, burning, foreign body sensation, mild pain, and general malaise which disables the patient. Other ocular disturbances, such as blepharitis and conjunctivitis, and rarely keratitis rosacea (and iritis rosacea) are also observed. The syndrome is well-known to occur with or without facial involvement. Rosacea faciei is characterised by appearance on the nose and cheeks of vascular dilatations, initially transient and later permanent, which may lead to a persistent telangiectasia.

The disease is of long duration, progredient in character, and a satisfactory therapy has not yet been devised.

Many theories (contradicting each other), concerning the aetiology of rosacea have been advanced. Bacterial pathogenesis,³ disturbances in the alimentary tract (hypochlorhydria),⁴ and secretory and angiomotor neurosis of a hereditary character⁵ have been suggested. A common assumption that the cause of keratitis rosacea is dietary abuse, exhaustion and irregularity in taking meals has been shown to be erroneous.⁶

Recently it has been suggested that riboflavin deficiency is the cause of keratitis rosacea, and may lead at the limbus to vascular dilatation such as is observed in keratitis rosacea. It has been pointed out, however, that the corneal vascularisation due to ariboflavinosis differs from that of keratitis rosacea.⁷ The efficacy of riboflavin treatment in keratitis rosacea has been doubted.⁸⁻¹⁰

The obscurity of the aetiology of keratitis rosacea is matched by the uncertainty of its therapy. It is generally recognised that treatment of this condition must be general and constitutional.¹¹ Local treatment (ichtho-zinc) is only palliative. General treatment has been attempted along different lines, *e.g.*, treatment of possibly present gastric malfunction, protein shock therapy, elimination of septic foci,¹¹ hormonal treatment with corticosterone,¹² and recently riboflavin.^{6, 7, 13}

Tests for presence of hormonal allergy have been performed in

6 cases of keratitis rosacea (3 men and 3 women; duration of disease from 4 to 20 years). All cases were refractory to accepted methods of treatment and showed gradual progress. The six cases of keratitis rosacea were tested with regard to several hormones with the following results: In all of them hypersensitivity to testosterone *only* was found (Fig. 1). Desensitisation treatment with small gradually increasing doses of the hormone produced in every case an improvement of the condition of the patient.

CASE 1.—G.A., female, aged 40 years, housemaid, regular menstrual periods, married for 16 years; three deliveries and one abortion. The father died of diabetes. Two children exhibited

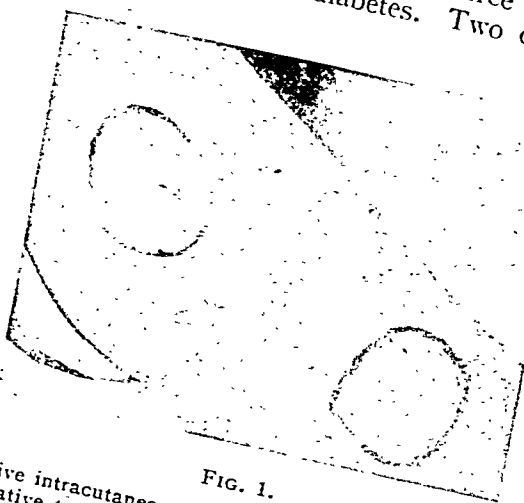


FIG. 1.

Left: Positive intracutaneous test with testosterone in oily solution.
Right: Negative test with oil control.

The first child, a 6 year old girl, is suffering from chronic urticaria of unknown aetiology. The second child, a 4 year old girl, suffers from spastic bronchitis. No other peculiarities were found in the patient's history. Rosacea faciei, with symptoms so severe that they were ascribed to lupus miliaris, set in 12 years ago. Concomitantly eye disturbances—blepharitis and keratitis rosacea—developed. The disease gradually became aggravated in the following years, but remissions occurred from time to time. The patient suffered very much from photophobia, burning and lacrimation which disabled her to a great extent. It is noteworthy that all these complaints subsided during pregnancy, but recurred in the period of lactation. There were no other symptoms that could in any way be related to the genital cycle.

Present condition. Extensive rosacea of the cheeks and nose and enlargement of the vessels, were seen giving the face a dark red

aspect; furthermore, bilateral blepharo-conjunctivitis was present, the vessels of the bulbar conjunctiva were markedly enlarged on both sides. The vessels around the limbus presented a garland-like appearance and partly encroached upon the cornea. Sub-epithelial infiltrates, some of them reaching the borders of the pupils, were observed, penetrating partly into the outer layers of the corneal parenchyma (Fig. 2). General examination revealed normal findings: Blood chemistry normal; urine normal; Wassermann reaction in blood negative; blood picture: erythrocytes

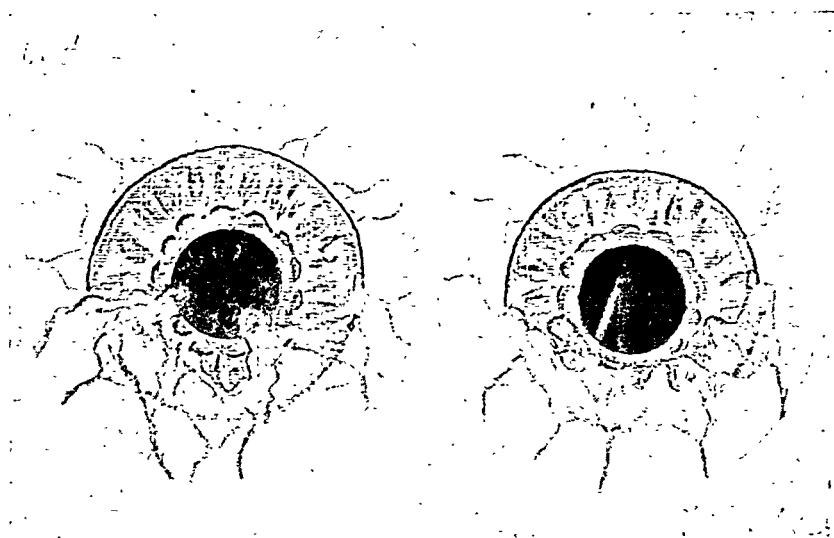


FIG. 2.

Left and right eye. patient (Case No. 1) suffering from keratitis rosacea before treatment. Note garland-like enlargement of the vessels around the limbus corneae.

4,840,000, slight leucocytosis—11,500, eosinophiles 3 per cent., blood sedimentation normal. The following intracutaneous tests for presence of hormonal allergy were carried out:—

- (1) ovarian series: oestradiol, oestrone, progesterone, pregnandiol;
- (2) adrenal series: corticosterone, testosterone, androsterone;
- (3) control series: cholesterol and oil.

The tests showed clearly a strong hypersensitivity to testosterone. Control tests with oil alone and cholesterol were negative. The desensitisation treatment consisted of subcutaneous injections of gradually increased doses of testosterone dissolved in oil (20 successive daily injections of doses ranging from 0.01 to 1 mg.). This therapy produced a definite improvement. The subjective

symptoms of the patient improved, the photophobia disappeared, there was no burning sensation, and the limbal vascular dilatation was considerably diminished. Four weeks after release from hospital, the patient was still free from her former symptoms, and only four weeks later she came to see us with a mild recurrence of the inflammatory symptoms. The corneal infiltrations extended from the nasal limbus to the borders of the pupils and showed characteristic degenerative changes. The patient was again given 10 injections of testosterone (0.1 to 1.0 mg.) and for consolidation of the treatment a tablet of 10 mg. was implanted subcutaneously.

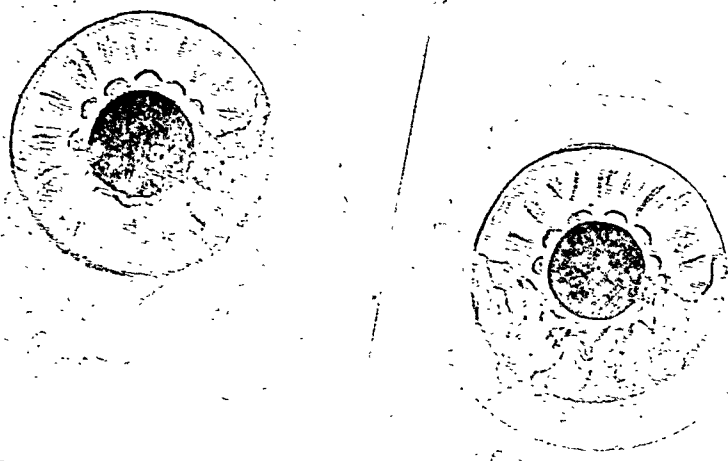


FIG. 3.

Same patient after desensitisation with testosterone. Note recession of the vascular engorgement.

This time the patient remained free from symptoms for a period of 3 months. So long an interval had not previously been experienced by the patient except during pregnancy. Then a mild inflammation set in. 25 mg. of testosterone were again implanted. Ten days after implantation an acute inflammatory reaction was present in both eyes which lasted for two weeks and was ascribed to a possibly excessive dose. Apart from this reaction the patient remained free from complaints. There was considerable improvement of the rosacea faciei. Vascular dilatation in the nose and cheeks regressed markedly, and the face became decidedly less red. Vascular injection of the conjunctiva bulbi and at the limbus diminished. The corneal opacities were unchanged after an observation period of one year (Fig. 3). Local therapy had been avoided as far as possible during this interval.

CASE 2.—K.W., male, aged 45 years, a policeman by occupation. Suffered for many years from chronic dyspepsia possibly as a sequel to chronic amoebiasis. For 6 years rosacea faciei with involvement of the eyes—conjunctivitis and keratitis rosacea. Two years before, the condition of the right eye became so serious that the patient had to be admitted to the hospital. Recurrent ulcers at the temporal border of the cornea were present at that time. Local treatment (cauterisation) and general riboflavin and vitamin A therapy were given. The recurrent inflammation of the eyes greatly hampered the patient at his work.

Present condition. Moderately severe rosacea faciei, bilateral blepharo-conjunctivitis, rosacea, particularly manifest in the left

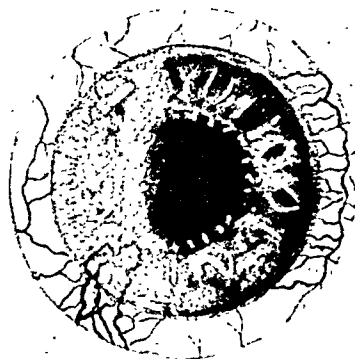


FIG. 4.

Left eye of patient No. 2 with typical keratitis rosacea before treatment. Note strong pericorneal injection of vessels protruding into the cornea.

eye as characteristic vascular dilations, partly forming a garland over the cornea; the superficial corneal layers were infiltrated (Fig. 4). Wassermann reaction in the blood was negative.

Intracutaneous test with hormonal allergens revealed a positive reaction only to testosterone. The tests were carried out with 0.1 mg. and smaller amounts of testosterone, a positive reaction being obtained even with 0.001 mg. of hormone.

Desensitisation treatment, consisting of a daily subcutaneous injection of a gradually increasing dose of testosterone, ranging from 0.001 mg. to 1.0 mg., was given for one month. The condition of the patient improved and the symptoms disappeared. After two months, mild recurrence of the symptoms was experienced. Ten more injections of testosterone in doses ranging from 0.1 mg. to 1.0 mg. were given, the treatment being consolidated by the

implantation of a pellet of 10 mg. of testosterone. Three months later a second pellet of 10 mg. was implanted. During the period of treatment only transient and mild manifestations lasting 2-3 days were observed. Eventually, a third pellet of 10 mg. was implanted. Local therapy was not employed during the whole time. The condition of the patient improved definitely, as there were no subjective symptoms and the vascular injection receded almost completely (Fig. 5). The rosacea faciei definitely improved as well. The patient, unhampered by any ailment, is again at work. The period of observation was twelve months.

CASE 3.—B.Z., female, aged 62 years, suffered for 20 years from keratitis rosacea accompanied by vascular dilatation on cheeks

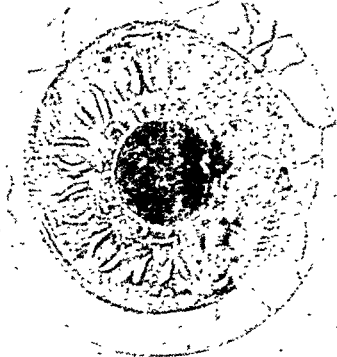


FIG. 5.

Left eye of same patient after desensitization with testosterone. Note disappearance of the vascular injection.

and nose. Recurrences of keratitis rosacea were so frequent that the patient was seriously handicapped in her work. The vessels around the limbus invaded the cornea. Sub-epithelial infiltrates reaching to the pupil area, in both corneae were particularly advanced on the right side. The patient complained of an intolerable almost constant sensation of burning, photophobia and of lachrimation. Intracutaneous tests with several hormonal allergens revealed a strong, positive reaction only in respect to testosterone. Acute inflammatory reaction developed in both eyes during the performance of the tests. Desensitisation treatment with gradually increasing daily doses of testosterone (from 0.01 mg. to 1 mg.) in a series of 20 injections administered subcutaneously was undertaken and finally a 10 mg. pellet of testosterone was implanted. Since this treatment, all the subjective symptoms as well as the

vascular injection receded almost completely. The vascular dilatation on cheeks and nose also greatly improved. During the three months following the conclusion of the treatment, the patient, for the first time in many years, was free from complaints.

The following three cases will be reported briefly as they have been under observation for a short period only; in the first of them desensitisation treatment has not yet been instituted.

CASE 4.—R.K., man, aged 42 years, mechanic by occupation, suffered for 5 years from rosacea faciei accompanied by keratitis rosacea. Failed to respond to different treatments, local and general, given during this period. Intracutaneous tests made with different steroid hormones gave a positive response only in respect to testosterone. For personal reasons, it was necessary in this case to postpone the treatment.

CASE 5.—The patient, a woman, suffered from rosacea faciei with keratitis rosacea. The symptoms were of long standing and resisted all earlier attempts at therapy. The intracutaneous tests with steroid hormones revealed hypersensitivity to testosterone only. Desensitisation treatment was instituted and produced a marked improvement. (A detailed report on this case will be published by Dr. Lass, Allergist, Hadassah Hospital, Tel-Aviv.)

CASE 6.—A.K., man, aged 65 years, a theological scholar, suffered for the past 17 years from keratitis and conjunctivitis rosacea with a mild facial involvement. Two months previously he suffered from a severe exacerbation of his ocular condition which did not yield to any local treatment. The intracutaneous tests with hormones revealed hypersensitivity to testosterone only. The treatment with gradually increasing doses of testosterone produced a striking improvement at the end of the first week, after which all inflammatory signs receded and the patient was able to resume his work.

Discussion

Allergy is considered to be the cause of a variety of eye diseases. In general the disturbance is ascribed to hypersensitivity of the ocular tissues to exogenous proteins. It has been assumed that human eye tissue can be allergic also to endogenous products of metabolism. In sympathetic ophthalmia, for instance, hypersensitivity to endogenous uveal pigment has been claimed.¹⁴ A positive cutaneous reaction to intradermal injections of uveal pigment extract has been demonstrated in cases of this kind.^{15, 16} Histologically cutaneous changes in this allergy resembled the histological changes in the eye.¹⁷ Allergy to endogenous lens protein has also been considered to be a cause of post-operative or post-traumatic endophthalmitis.^{18, 19} In our cases it seems necessary to assume the presence in the ocular tissue of a state of allergy

to endogenous hormones. It is of interest that in these cases the endocrine allergic reaction was not directed to protein hormone substances but to steroid hormones of a relatively simple structure. It is remarkable that in the six cases (three men and three women) examined by us the responsible hormonal allergen was always testosterone. This hormone is produced not only by Leydig cells of the testis, but also by adrenal tissue, and this circumstance probably explains the occurrence of an allergy to this substance in mature women. Our findings, pointing in one direction, of course do not exclude the possibility that other aetiological factors may also play a rôle in keratitis rosacea.

Treatment of hypersensitivity to endogenous hormones consists of specific desensitisation by daily subcutaneous injections of the offending specific hormonal allergen, in small and gradually increasing doses and in the present cases with testosterone. All cases showed definite improvement under this therapy. However, mild and transient recurrences were observed after a long interval following the completion of the treatment course. Regular repeated implantations of pellets containing 10 mg. (or may be even less) of the allergen at intervals of 3-6 months are therefore indicated as a means of consolidating the therapy. The initial doses of testosterone employed in desensitisation should be very small, never exceeding 0.1 mg., and the increase should be gradual. These precautions seem to be necessary since focal reactions have been observed after administration of larger doses, as witnessed in our first case. Similar precautions should be observed in pellet implantations.

Therapeutic results in keratitis rosacea must, as a rule, be interpreted with great caution, as spontaneous remissions occur frequently. Nevertheless, the results described above lead us to the conclusion that the treatment was specific. The subjective symptoms improved and the objective signs, the limbal and corneal vascular dilatation, also changed for the better. All patients were relieved, symptom-free periods of equally long duration had not occurred in the absence of treatment or after other treatments. Recurrences were noted in the form of slight vascular injection which, however, receded promptly after a second course of desensitisation with testosterone. An effect on the permanent degenerative corneal changes could of course not be expected.

Endocrine allergy affords a possible explanation for the unknown aetiology of certain diseases. While in some of them the offending hormonal allergen may vary (e.g., in urticaria positive reactions were found with regard to oestrone, testosterone and corticosterone) it is noteworthy that in our small group of cases of keratitis rosacea all showed allergy to one and the same hormone,

Proceeding from the assumption that the ocular tissues are

capable of allergic reaction to endogenous hormones, other eye diseases of obscure aetiology and long duration, and in which an allergic basis could be suspected, have been studied from this point of view (chronic, periodically recurrent conjunctivitis and undetermined cases of keratitis superficialis as well as other diseases²). The results obtained in these cases owing to desensitisation treatment with the respective hormonal allergens have been encouraging.

Summary

1. Six patients with keratitis rosacea associated with rosacea faciei (3 men and 3 women) of long duration which had proved refractory to the usual methods of treatment, were examined for hypersensitivity reactions to endogenous hormones.

2. In all cases allergy to testosterone was demonstrated by positive skin reactions to intracutaneously injected testosterone. Other hormones (oestrone, oestradiol, progesterone, pragnandiol, corticosterone, insulin, gonadotropin) gave negative reactions.

3. Desensitisation treatment by a course of subcutaneous injections of testosterone in gradually increased doses and implantation of pellets containing 10 mg. of testosterone propionate produced satisfying results.

4. Testosterone gave relief only when given in low, gradually increased doses; administration of large doses may cause a severe exacerbation.

5. The finding of allergy to endogenous testosterone in a small number of cases of keratitis rosacea does not exclude the possibility:—

- (a) that other endogenous hormones may act as allergens in some cases;
- (b) that other causative factors are involved in the aetiology of keratitis rosacea.

The results achieved were as follows:—

- (a) Complete and prompt cessation of the subjective symptoms, photophobia, lacrimation and sensation of burning in the eyes.
- (b) Considerable improvement or disappearance of the limbal and corneal vascular dilatation, as well as a marked improvement of the facial rosacea.
- (c) Mild recurrences in some instances which promptly improved after administration of a second course with testosterone treatment.

We are indebted to Prof. A. Feigenbaum, Head of the Ophthalmological Department, Rothschild Hadassah University Hospital,

for his help and keen interest in this paper, and to Dr. B. Mitterstein, First Assistant of the Ophthalmological Department. We are furthermore indebted to Dr. Hilde Rosenburger for the drawings in Figures 2-5.

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SOME OBSERVATIONS ON THE SYMPTOMATOLOGY AND DIAGNOSIS OF CASES OF PROPTOSIS*

BY

PROFESSOR HANDOUSA BEY

CAIRO

I HAVE had to examine and treat a large number of cases of proptosis, most of them referred to me by the various ophthalmic centres in Egypt. Study of the records of these cases reveals some valuable points worthy of publication.

The types of cases that I have come across, fall into the following categories:—

- 1.—Cases of Extra-Orbital Causation.
- 2.—Cases of Intra-Orbital Causation.
- 3.—Cases due to disease of the bony wall of the orbital cavity.

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1.—Extra-orbital cases

These were mainly of nasal origin and inflammatory in nature. The frontal and ethmoid para-nasal sinuses were the main primary sites and usually the spread took place in an acute case or during an acute exacerbation on top of a chronic one.

Nasal trauma, mucocoeles, pyocoeles and neoplasms, were not uncommon causes.

Maxillary sinus infection in my cases has never been seen to give rise to proptosis unless the related anterior ethmoid is infected too, a point indicative that the infection has spread from the latter.

Nasal neoplasms causing proptosis were mainly malignant, affecting the ethmoid and maxillary regions. They were either carcinomata, sarcomata, or endotheliomata, with 4 cases of osteoclastomata and 3 cases of adamantinomata.

Nasal neoplasms of simple nature causing proptosis were mainly osteomata. I had only four cases of ethmoid fibromata and two ethmoid hæmangiomata.

Nasopharyngeal tumours, whether simple or malignant have been commoner causes of proptosis than usually supposed, a point that stresses the importance of examining the nasopharynx in cases of proptosis of vague causation.

2.—The Intra-orbital category

I included under this heading all the cases in which the causative lesion has started inside the orbital cavity.

These were 31 in number, grouped as follows:—

1. *Inflammatory*—(a) One case of amyloid change in the orbital tissues round the globe, most probably secondary to chronic suppuration around the teeth and in the maxillary antra.

(b) One case of syphilitic change in the orbital contents on both sides, associated with enlargement of the parotid and submaxillary salivary glands simulating Mikulicz's disease.

2. *Cysts*—(a) Intra-orbital dermoids related to the upper eye-lid region.

(b) Retention cysts of the lacrimal gland.

(c) Haemorrhagic cyst of the lacrimal gland.

(d) Parasitic cysts—2 cases of hydatids. One lying inside the muscle cone and one lying outside it related to the orbital floor.

3. *New-growths*—These were the commonest in this category, (23 cases out of 31), 6 were simple, in the nature of fibromata, fibro-lipomata, rhabdo-myoma and lymph-angiomata: The malignant types were sarcomata, carcinomata, endotheliomata and one case of xanthoma. The carcinomata and endotheliomata were all arising from the lacrimal gland, the xanthoma was most probably arising from the tendon of the superior oblique muscle.

3.—Cases due to a disease of the bony wall of the orbital cavity

These were the least common among my group of cases and may be grouped as follows:—

1. *Traumatic*—One case of old fracture of the left maxilla that resulted in a large gap in the floor of the left orbit with the formation of an extensive arterio-venous varix, which pressed on the left globe and induced proptosis on stooping down.

2. *Inflammatory*—(a) Pyogenic, of the nature of osteomyelitis (acute and chronic) of the orbital wall.

(b) Tuberculous abscesses, and gummatous formations of the orbital wall.

3. *New Growths*—I have one case of capillary angioma of the frontal bone causing right proptosis, a very rare condition.

Study of the symptomatology of these cases shows that in spite of varying degrees of proptosis and eyeball deviations, diplopia is exceptionally complained of. I thought at first that this may be because the patients were more concerned with the bulge of the eye but I directed their attention while being under investigation in hospital to the possibility of seeing double; still this was not complained of except very rarely. Twenty successive patients were carefully tested, only in two diplopia was reported.

As regards diagnosis of the underlying cause of proptosis, I should like to stress that pitfalls with serious consequences are very likely unless careful history taking, systematic local and general examination is carried out in a routine manner, assisted by the various radiological and laboratory investigations. Many demonstrative examples can be given; I have among my cases, patients that were referred to me as having sarcomata of the orbit, with local clinical signs, suggestive of malignant disease, but simple careful history taking made me doubtful and with systematic investigation, these cases proved to be inflammatory. Inflammatory masses in the orbit may closely simulate malignant disease in many clinical aspects.

I have met with few but very valuable points in radiological study of my cases which are worth special consideration. These are:—

1. The pathological lesion in middle and posterior ethmoiditis may spread towards and into the orbital cavity giving rise to proptosis at the same time not presenting any clinical evidence intra-nasally. Even in some of these cases X-ray examination in the classical positions may prove to be negative. However, if this is repeated in various other positions, definite radiological signs may be found to indicate the site of the disease in the ethmoid. I have had two such cases which were considered clinically and radiologically negative for sinus disease and the cause of the proptosis was not then determined. Interest and patience in repeating the X-ray examination cleared up the diagnosis of ethmoiditis.

Fig. 1. is an X-ray which is very definite of left ethmoiditis with thickened bone was only arrived at after repeated negative trials in a case of proptosis.

2. Comparative X-ray study of both orbits has indicated or defined the site of the underlying cause of proptosis in many a case of obscure origin.

I shall stress here only two points—

(a) The presence of an X-ray shadow of the tumour mass.

(b) The presence of an X-ray shadow of the primary orbital dilatation.

(a) With appropriate technique not only bony but also soft tissue masses can be demonstrated deep in the orbital cavity. I have cases in which the radiologist has succeeded even in demonstrating the shadows of both eye-balls in normal cases.

This fact was made use of in demonstrating tumours lying far back in the orbital cavity, not palpable or demonstrable otherwise, and is always worth a trial.

X-ray shadows of bony masses are well known; shadows given by soft tissue masses have been of two types: dense and light.

Dense shadows when present were always diagnostic of the underlying cause of proptosis. Fig. 2 is a demonstrative example of such a dense shadow. This patient is an old exophthalmic goitre case in which appropriate treatment resulted in great diminution of his exophthalmos with fall of his B.M.R. from (+45 to -7). It was noticed about one year later that his right eye-ball was again proptosing gradually. Investigations were negative as to the cause, but with careful radiological investigation and comparing both orbits a shadow could be seen in the right orbit (Fig. 2). This on exploration proved to be a rhabdomyoma, removal of which resulted in cure.

Light shadows show as veiling particularly over the orbital periphery. They were always found to correspond with the congested and oedematous orbital tissues and so they demonstrate secondary manifestations and not the primary cause of the proptosis. Fig. 3 is an example of such a shadow.

(b) Orbital dilatation has been seen to be produced by many extra- and intra-orbital lesions. For descriptive purpose may I term the former secondary and the latter primary dilatation. In my experience there has been no difficulty in differentiating one from the other.

As an example of secondary dilatation I refer to the X-ray (Fig. 4), in which the dilatation has been demonstrable in the film to be due to a communication between the orbital cavity and the dilated ethmoid. On the other hand X-ray (Fig. 3) shows simple orbital dilatation which is not due to any communication with neighbouring cavities, hence the term "primary dilatation."

Primary dilatation has been found consistent with the presence of an intra-orbital lesion and fully justifies exploration.



FIG. 1.



FIG. 2.



FIG. 3.

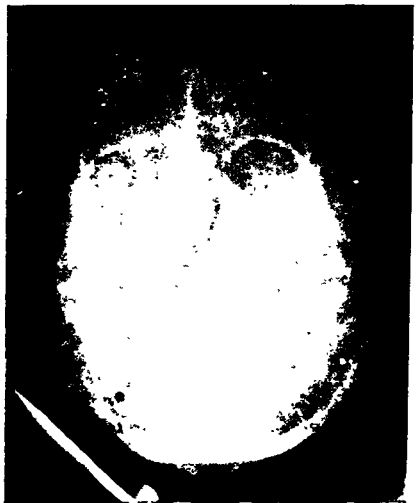


FIG. 4.



FIG. 5.



FIG. 6.

This sign was positive in 16 cases out of 21, and was the only evidence of intra-orbital tumour, a case in which a deep fibroma was found and removed on exploration.

It was negative in the following cases, all of which had other diagnostic signs:—

1. One case of osteoma of the ethmoid bone which was lying superficially and was palpable.

One case of myoma.

Both these gave definite X-ray shadows that were diagnostic.

2. One case of fibro-lipoma.

One case of ? Mikulicz's disease.

One case of endothelioma of the lacrimal gland.

In these three a mass could be palpated.

So these radiological findings may be of immense diagnostic value in obscure cases and justify exploration if need be.

Therapeutic tests: have been to me of great value in certain cases and so I feel they deserve trial if need be.

I had a case of a palpable mass in the left orbit firmly adherent to the bone and completely fixing the globe. The clinical signs were very suggestive of a new growth. This patient had an empyema of the left maxillary antrum with left ethmoiditis. On repeated punctures the eye condition improved considerably in 3 weeks. (Compare Fig. 5 and Fig. 6). With sinus treatment the patient was cured. This case demonstrates clearly the value of the therapeutic test (antral puncture).

There are other cases who had a positive Wassermann reaction—anti-syphilitic treatment resulted in resolution of the eye condition and saved major surgical intervention.

Exploration and biopsy has to be finally resorted to in obscure cases.

It has been my routine in any case where the lesion looked malignant on exploration to have a biopsy done before attempting to do radical operations. My case of amyloid disease demonstrates the wisdom of this conservatism. In this case all the appearances during exploration were suggestive of malignancy. If radical treatment was attempted straight out he would have lost the remaining bit of vision in that eye, a vision that is very much needed to him as he has a nebula on the opposite cornea.

Even if, on exploration, malignancy is certain, biopsy alone should be done as it determines the type of malignant disease present; a point which is of the utmost help in planning the line of treatment.

Cyst puncture: As a diagnostic aid has been advised and carried out by many. I am of opinion that it should not be freely done, as aspiration of the cyst contents not only makes the operation of complete cyst removal difficult, but also the procedure of puncture in itself may spread the disease in cases of hydatid cysts and breaking down malignant tumours.

I thank Prof. Sorour Bey and Dr. Hashem for their work on the pathology of my cases. I feel greatly indebted, too, to our expert radiologists, particularly Dr. Nissim Abou Saif and Dr. A. Marey for their valuable radiological study of these cases. I would like to express my deep gratitude to Dr. A. Barbary and Dr. Louis Labib who have assisted me in the operative work and have carried out a large part of the investigations, also to Dr. Ibrahim Mossalem, who has done the drawings, and to Sayed Ibrahim Eff, who has typed this paper.

THE INFLUENCE OF INTRA-OCULAR PRESSURE ON THE RATE OF DRAINAGE OF AQUEOUS HUMOUR. STABILIZATION OF INTRA-OCULAR PRESSURE OR OF AQUEOUS FLOW? * †

BY

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I.—Introduction

THE problem of the stabilization of intra-ocular pressure has aroused much interest. There are theories postulating reflex regulation of some kind or other. These have been critically examined elsewhere (Bárány, 1946 *a*). On the other hand, there are two theories, namely those of Duke-Elder and of Friedenwald and Pierce, which put the burden of stabilization on the mechanism of aqueous outflow.

Based on his elegant determinations of intra-scleral venous pressure in the dog, Duke-Elder (1926) has proposed a "safety-valve action" theory of the aqueous drainage mechanism. According to this theory, the pressure relations between the anterior chamber and the drainage channels are such as to prevent any flow of aqueous at normal intra-ocular pressure. When the intra-ocular pressure is raised, however, the pressure relations are reversed and become favourable for drainage of the aqueous. It is obvious, that any drainage mechanism which permits an increase of aqueous outflow with increasing intra-ocular pressure will, in a certain sense, tend to stabilize the intra-ocular pressure. The efficiency of a mechanism in stabilizing intra-ocular pressure depends on the slope of the curve relating aqueous flow to pressure. If the mechanism is efficient, even a small change in pressure will cause a large change

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† The radio-active sodium used in the experiments was kindly furnished by Prof. Manne Siegbahn, Research Institute for Physics, Stockholm.

in aqueous outflow. The "safety-valve" mechanism of Duke-Elder is one way in which this could be realised without necessitating a large flow of aqueous under basal conditions.

The application of Duke-Elder's theory to other species has been criticised, however, from an anatomical point of view by Troncoso (1942) after a thorough study of the vascular apparatus of the drainage angle. In view of the conclusive demonstration by Ascher (1942) and independently by Goldman (1946) of a continuous aqueous outflow in man and the proof of a considerable outflow under completely physiological conditions in the rabbit's eye by Kinsey and Grant (1942) the theory must probably be modified at least for these species.

It might conceivably be possible to retain the gist of the "safety-valve" theory by restating it as saying that the intra-ocular pressure is stabilized by means of an overflow mechanism.

If the drainage mechanism is to function as an overflow arrangement, which seems to agree with the ideas of Friedenwald and Pierce (1932), two conditions have to be fulfilled. First, the pressure in the drainage channels, as determined by the intra-scleral circulation, must be—if not actually higher than the intra-ocular pressure—at least only a little lower. Second, the resistance to aqueous flow from the anterior chamber to the drainage channels must be very low. If the drainage mechanism acts as an overflow arrangement, then the normal, not inconsiderable flow of aqueous is the result of a small driving force and a small resistance and even a small change in intra-ocular pressure will cause a large relative change in driving force and a large relative change in aqueous flow.

Since at present no means are known for measuring the pressure in the actual drainage channels nor for determining the resistance to flow from the anterior chamber under physiological conditions, the efficacy of the drainage mechanism as a pressure stabilizer can only be tested by direct measurement of the influence of intra-ocular pressure on the rate of aqueous flow.

This has been tried before by Niesnamoff (1896) and by Friedenwald and Pierce (1932). These authors introduced cannulas into living eyes and measured the rate at which normal saline entered the eye at different pressures. For a rabbit's eye Niesnamoff found an inflow of 1 mm.^3 per minute at 25 mm. Hg and of 5 mm.^3 per minute at 33 mm. Hg. For dogs, Friedenwald and Pierce found an inflow of 5-10 mm.^3 per minute at a pressure not more than 5 mm. Hg over the normal level of the anaesthetised dog. In Niesnamoff's rabbit, the intra-ocular pressure was obviously a little lower than 25 mm. Hg, probably about 23 mm. Hg. Thus, a pressure increase of about 10 mm. Hg above normal brought an increase of aqueous flow by about 5 mm.^3 per minute. Under completely physiological conditions, the rate of flow in rabbits is

about 4 mm.³ per minute (Kinsey and Grant, 1942). On a relative scale, therefore, Niesnamoff found an increase of 125 per cent. in aqueous flow caused by an increase of 43 per cent. in intra-ocular pressure. A similar calculation cannot be made for the data of Friedenwald and Pierce because we have no corresponding information about the rate of flow of aqueous in the dog under completely physiological conditions.

However, the results quoted above are open to criticism because they were obtained with a cannula in the anterior chamber of anaesthetized animals, that is on eyes under definitely unphysiological conditions. Moreover, the rabbit data derive from one experiment on one eye only and there might be species and individual differences. It therefore seems justified to take up the question anew. I have therefore used a new method to obtain an idea of how rapidly the aqueous outflow changes with a change in pressure in eyes which have not been subjected to any trauma whatever. Unfortunately, the method does not permit a study of the functional relation over a large range of pressure but only gives the slope of the curve in the vicinity of the normal equilibrium. However, this is the most interesting region from the point of view of physiological regulation.

II.—Experimental part

The principle of the method used is the following:

Under equilibrium conditions the composition of the aqueous is constant. This means that per unit of time, the same net amount of any substance as enters the anterior chamber will have to leave it by means of aqueous outflow or by diffusion. If diffusion plays no part the equilibrium condition is

Net rate of entrance E_0 of substance =

(concentration C_0 of substance) \times (rate of flow of aqueous F_0).

If we now change the equilibrium level of the intra-ocular pressure of an eye by some non-traumatic intervention, the corresponding rate of aqueous drainage F_1 can be obtained from the relative change in rate of entrance of a substance and the relative change in its equilibrium concentration.

$$\frac{F_1}{F_0} = \frac{E_1/E_0}{C_1/C_0} \quad (1)$$

Thus, the experimental problem consists in finding a suitable non-traumatic way of changing the intra-ocular pressure and a suitable substance. Then the related changes in intra-ocular pressure, equilibrium concentration and net rate of entrance of the substance are determined.

In the experiments to be described, the change in equilibrium levels was brought about without touching the eye proper by unilateral closing of the common carotid artery in rabbits. A very large

number of experiments on unanaesthetized animals have shown that, on an average, this reduces the mean blood pressure in the ophthalmic arteries by about 30-40 mm Hg from the normal level of about 100 and the intra-ocular pressure by about 4 mm Hg from the normal level of about 30. The pressures assume new levels which do not change appreciably from 10-20 minutes after closing of the vessel to 24 hours after the operation (Bárány 1946a, d, 1947b).

Carotid closure may appear to be a rather indirect way of changing the intra-ocular pressure. It has the quite invaluable advantage, however, of leaving the other eye unchanged and usable as a control.

The test substance used for the studies was the radio-active sodium isotope Na (24). As Kinsey and Grant (1942) have shown, there is no significant diffusion exchange of sodium between aqueous and blood, sodium enters by secretion and leaves by a process of flow. Consequently, this substance is especially suited for our purposes.¹

Aqueous samples can be obtained under topical anaesthesia but it is much easier and safer to use general anaesthesia. The question then arises, whether the drug used, allyl-isopropyl barbituric acid, Numal Roche, might influence the relevant relations between levels of concentrations and pressures. In order to avoid possible pitfalls here, some of the experiments to be described were made with intra-peritoneal administration of Numal. In these cases the animals had been under the influence of the drug for up to two hours when the aqueous was withdrawn. In other experiments, the drug was given intravenously only 5 minutes before the corneal puncture. There was no noticeable difference between the results. The pressure effect

¹ The fact that radio-active test substances in the body fluids can be detected by means of counter arrangements outside the body suggests the following possibility for studying the rate of aqueous exchange in the intact eye: By means of a suitable time schedule of injection (if necessary controlled by continuous observation of the plasma concentration with a counting rate meter) a practically constant plasma level of a test substance could be maintained from the very start. The test substance preferably should be a radiator of not too penetrating corpuscles. A counter arrangement subtaining a small "visual angle" and equipped with suitable filters could then be arranged to measure radiation practically exclusively from the cornea and the superficial layers of the aqueous. The accumulation of the test substance in these two compartments could thus be followed. The fluid exchange of the cornea is slow and the total electrolyte content relatively low. Therefore, the initial slope c of the accumulation curve would certainly depend almost exclusively on the rate of accumulation of radio-active substance in the aqueous and thus be a measure of the rate of entrance of the test substance in arbitrary units. After a certain interval, the aqueous concentration would have reached an equilibrium level. If the corneal concentration were still sufficiently low, the radiation from the eye at this juncture would express the equilibrium concentration c of the substance in the same arbitrary units as the initial slope c . The quotient e/c would then be a measure of the rate of aqueous exchange, showing directly how large a fraction of the test substance is exchanged per unit of time. It is possible that the amount of radiation coming from the substance of the cornea will no longer be negligible when the aqueous equilibrium is reached. This would hamper the application of the method to human eyes, if no method is found for isolating the corneal radiation. In animal experiments, the activity of the cornea could be determined separately after removal.

The author is engaged in the construction of equipment necessary for experiments along these lines.

of the carotid closure has not been studied as extensively under Numal as in the unanaesthetized state, because it is much more difficult to tonometrize under Numal. The rabbits often develop considerable nystagmus and with our standardized technique, the flaccid animals are more difficult to handle. Enough measurements have been made, however, to allow the statement that on an average carotid occlusion gives about the same pressure drop under Numal as in the unanaesthetized state. The normal intra-ocular pressure is about 5 mm Hg lower under Numal.

The equilibrium concentration of $\text{Na}^{(24)}$ in the aqueous after carotid occlusion was determined in 12 rabbits. One common carotid was ligated and an intra-peritoneal injection of $\text{Na}^{(24)}$ given within 2 hours afterwards. After about 15 hours, an ample time for equilibration, samples of aqueous were drawn from both eyes and the activity of the samples determined with a conventional counter arrangement, with a capacity well above the activity of the samples. At least 2-3000 impulses were counted, the counting error thus is about 2 per cent. Tonometry of the eyes was not performed in order to avoid every trauma.

Some of the animals which had received Numal intra-peritoneally needed a few supplementary drops of ether and were moreover given one drop of 0.4 Diocain Ciba topically immediately before the paracentesis. Details of technique have been described elsewhere (Bárány 1947c). The last animal in the intra-peritoneal group died a few minutes before the aqueous was aspirated. It is included in the table all the same, since there is little reason to expect the equilibrium to change during the first minutes after cessation of breathing.

The result is summarized in table I.

The table shows that there was almost no difference between the sodium content of the aqueous on the side of the closed carotid and that of the control eye. The mean ratio is 0.99. The kind of anaesthesia obviously played no rôle.

There seems to be only one systematic source of error which could play a part in this result, namely contamination of the aqueous with plasma. It is well known that the reduction in intra-ocular pressure caused by a corneal puncture leads to a capillary dilatation and the formation of plasmoid aqueous. When drawing aqueous samples one tries to avoid contamination with newly formed plasmoid aqueous by avoiding contact with the iris, by not emptying the anterior chamber completely, by using only very gentle aspiration and by working quickly. All the same, some slight admixture of plasma might easily occur. We know that at equilibrium in the normal eye, the sodium concentration in the aqueous is about 90 per cent. of that in the plasma. Assume that the aqueous withdrawn from a normal eye is contaminated with 10 per cent. of plasma. A simple calculation shows that this will increase the sodium content of the aqueous by 1 per cent.

TABLE I

The influence of unilateral carotid occlusion on the equilibrium concentration of radiosodium in the aqueous humour.

All activities are corrected for radio-active decay. They have been made comparable by multiplication by the body weight of the animal, because all animals received the same dose of radiosodium. The blood pressures were obtained by means of an ear capsule about 24 hours after the withdrawal of aqueous and about 40 hours after ligation of the carotid.

Animal Nr	Body weight kgm.	Aqueous, counts per minute per 0.1 ml, multiplied with body weight			Mean blood pressure, Occluded Control	Anaesthesia, Numal ml/kgm
		Occluded	Control	Occluded Control		
10	2.0	766	790	0.97	0.49	0.6 i.v.
12	1.9	1010	1065	0.95	0.53	0.6 i.v.
13	2.0	915	928	0.99	0.48	0.6 i.v.
14	2.1	767	734	1.04	0.57	0.6 i.v.
15	2.1	820	835	0.97	0.60	0.6 i.v.
16	1.7	795	843	0.94	0.43	0.6 i.v.
18	2.3	798	829	0.96	0.71	0.65 i.p.
19	1.9	880	911	0.97	0.48	0.65 i.p.
20	2.2	959	943	1.02	0.69	+0.15 i.v. 0.65 i.p.
21	2.1	890	870	1.01	0.48	0.65 i.p.
23	2.5	790	809	0.98	0.56	0.65 i.p.
24	2.5	848	815	1.04	dead	0.65 i.p.
				0.987	0.547	

Now, as is well known (Wessely 1908), plasmoid aqueous is less easily formed on the side of the closed carotid, where the blood pressure is much lower. Therefore, on an average this source of error will tend to increase the sodium concentration predominantly in the control eye. Some part of the slight difference between

experimental eye and control eye found might depend on this source of error.

This result, that carotid occlusion hardly or not at all changes the equilibrium concentration of sodium in the aqueous is in excellent agreement with similar experiments previously made concerning the total osmotic pressure of the aqueous (Bárány 1946*b*, 1947*d*). In 29 experiments on 22 animals, using the Hill-Baldes' thermo-electric vapour tension method, it was found that carotid occlusion changes the osmotic pressure of the aqueous by less than a fraction of one per cent. As sodium chloride is responsible for the absolutely dominant part of the osmotic pressure of the aqueous, the result can hardly imply anything other than that carotid occlusion does not change the sodium chloride concentration.

Thus we have found that in equ. (1) $C_1 \cong C_0$. But then the expression degenerates into

$$\frac{F_1}{F_0} \cong \frac{E_1}{E_0}$$

It is interesting to note that, because $C_1 = C_0$, the same relation would hold even if sodium had entered by diffusion instead of secretion.

Thus, the constancy of the sodium concentration shows that the relative change in aqueous outflow caused by carotid closure must be very nearly equal to the relative change in rate of entrance of sodium.

How large, then, is the change in rate of entrance of sodium caused by carotid occlusion? With the aid of the result just arrived at, this can be deduced from previous measurements by the author, made for a different purpose (Bárány 1946*c*, 1947*c*).

In these experiments, Na (24) in the form of 5 ml. of normal saline was injected into rabbits intra-peritoneally. One carotid of the animals had been closed a few hours earlier. After 40-60 minutes, aqueous samples were withdrawn and the activities measured. At this stage, the Na (24) content of the aqueous is still far below that of the plasma. The figures obtained would therefore be almost direct measures of the rates of entrance of Na if the rate of aqueous outflow had been constant. But, as carotid occlusion reduces the intra-ocular pressure and the rate of aqueous drainage, any reduction in rate of entrance caused by carotid occlusion will be partly compensated for by the concomitant reduction in aqueous drainage. Consequently, a correction factor has to be applied. This can be derived as follows:

The experiments were made under the same conditions as these of Kinsey, Grant, Cogan, Livingood and Curtis (1942) on which the theoretical treatment of Kinsey and Grant (1942) is based. Therefore, equ. (10) of these latter authors which fits their sodium data should be applicable to ours too. The equation is

$$c_2 = -k_2 \cdot c \left\{ \frac{1}{k'} \left(1 - e^{-\frac{k' t_1}{V}} \right) - \frac{1}{k' V - k'} \left(e^{-\frac{k' t_1}{V}} - e^{-k_1 t_1} \right) \right\}$$

Here, c_2 is the radio-sodium concentration in the aqueous,

c the equilibrium concentration in the plasma,

k_2 the co-efficient of rate of entrance,

k' the co-efficient of rate of exit by aqueous flow,

$1/k_1$ the time constant of the equilibration process between the intra-peritoneal depot and the blood,

t_1 the time after intra-peritoneal injection of the sodium salt,

V the volume of the anterior chamber.

The numerical values of the constants are as follows: k' was found by Kinsey and Grant to be about 4 mm.³ per minute in the normal rabbit's eye, V is about 250 mm.³ and t_1 in our experiments was about 50 minutes. For k_1 we can get a value from the plasma concentration curves of Kinsey *et al.*, which show full concentration after 20-30 minutes. This time thus is about 3 time constants long and k_1 consequently is about 0.1-0.2.

Now, what we want to know is how a proportionally equal reduction in the rates of entrance k_2 and of exit k' influences the concentration c_2 as compared with the other eye of the same animal where k_2 and k' are uncharged.

If the above numerical values are inserted and the expression is evaluated by series expansion including the fourth order terms the following figures are obtained:

Reduction of rates k_2 and k' in per cent. of the normal 10 20

Reduction of concentration c_2 in per cent. of the normal 7.3 15

This result is obtained with $k_1 = 0.1$. If the upper limit $k_1 = 0.2$ is used instead, the figures become 7.0 and 14.5. On the other hand, if the fifth-order terms are included, the figures increase by a few tenths of one per cent.

The result of the calculations shows, that under our conditions of experiment, the relative reduction in the rate of entrance of sodium is reflected in the scale of 3:4 in the reduction in concentration 50 minutes after the injection. This knowledge allows us to make use of the concentration ratios between experimental eye and control eye obtained in the previous study. These ratios were

0.71, 0.92, 0.93, 0.93, 0.96, 0.98, 1.03, 1.22

The extreme values are without doubt the result of contamination with plasma, which in these experiments had an activity at least twice as high as the aqueous. The mean ratio is 0.96 and the median 0.95. The reduction in concentration caused by carotid clamping is thus about 5 per cent. or probably less because of the systematic error discussed before. Applying the correction factor to this value, we find the reduction in rate of entrance of sodium to be about 7 per cent. And now we have the answer to our problem, because in view of the constancy of the equilibrium concentration, this figure must also very nearly represent the reduction in aqueous outflow caused by carotid occlusion.

The remarkable thing about this figure (7 per cent.) is that it is so small. Even if various errors of estimation have co-operated to give too small a value, this result certainly does not support the idea of an overflow mechanism in the intact rabbit's eye. An intra-ocular pressure drop of 10-15 per cent. should have given a much more

pronounced reduction in aqueous outflow if an overflow mechanism had really existed. The discrepancy is even more striking if one considers the reduction in driving hydrostatic pressure head instead of intra-ocular pressure. According to Seidel (1923), the pressure in the episcleral veins is about 9 mm. Hg in the rabbit. Thus, the hydrostatic pressure in the aqueous drainage channels cannot be less than, say, 10 mm. Hg. The pressure head for aqueous outflow is thus certainly less than 20 mm. Hg in the normal eye and carotid closure causes a reduction amounting to certainly more than 20 per cent. Still there is only a small reduction in aqueous outflow.

III.—Theoretical part

A discrepancy of the magnitude found between the change in aqueous flow and in the pressure head must have an explanation. There seem to be two possibilities.

The first is that in our experiments, the rate of aqueous outflow was influenced, not only by the change in intra-ocular pressure but also by the change in arterial blood pressure as such. If the pressure in the drainage channels were directly dependent on the arterial blood pressure to any appreciable degree, a reduction in arterial pressure could conceivably increase the pressure head between the anterior chamber and the drainage channels and thus partly compensate for the reduction in intra-ocular pressure. It seems improbable, however, that this mechanism is very important. The drainage channels are venous vessels, the pressure of which is only dependent to a very slight extent on the arterial blood pressure. In so far as there is a small dependence, however, this mechanism could explain part of the discrepancy.

The other and more probable explanation is that the force which causes the outflow of aqueous consists of two parts, one of them a hydrostatic part which changes directly with the intra-ocular pressure and the other one an osmotic part, which is more or less constant.

The idea that osmotic forces play a part in the mechanism of Schlemm's canal originates from Friedenwald and Pierce (1932). Troncoso (1942) assumes that the drainage vessels of the sub-primate eyes, the trabecular veins, contain flowing blood which osmotically attracts aqueous humour. In animals, Friedenwald and Scholz (quoted Friedenwald 1940, 1942) have demonstrated "plasma and some red blood cells in the vessels which formed the base of the sclero-corneal trabeculum" after freezing the anterior segment of the eye with liquid air. The present findings indicate the existence of a constant absorptive force besides the changing hydrostatic one. There is thus every reason to consider how the mechanism of aqueous drainage will behave if the movement of the aqueous is the result of a combination of the two forces working in the same direction. The

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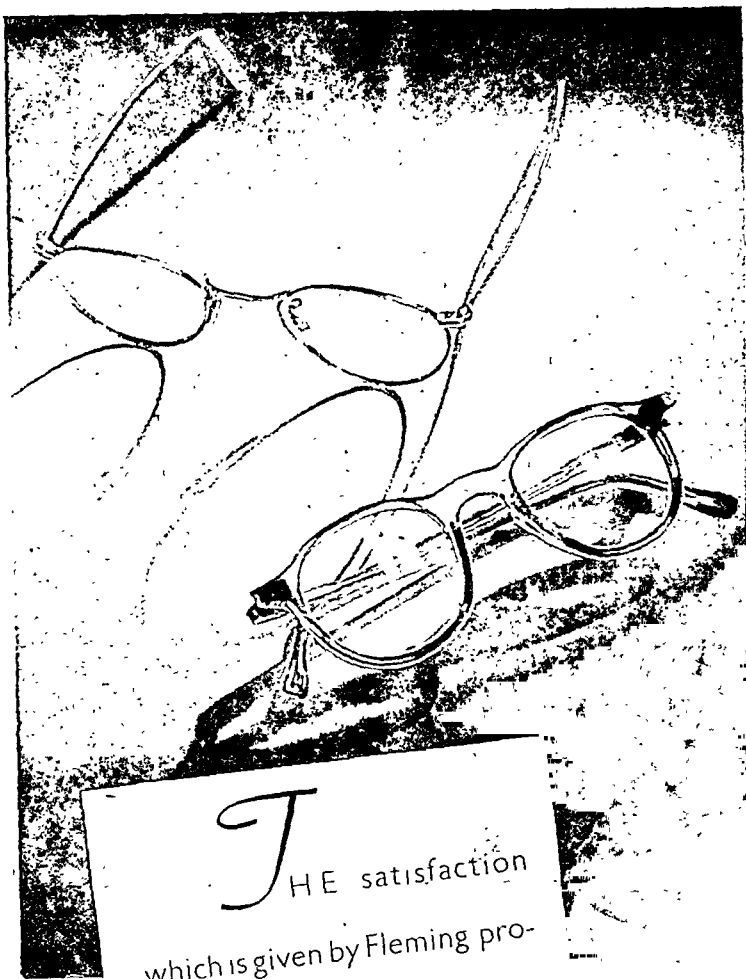


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histological findings of Friedenwald and Scholz indicate that attention should be given to the possibility of a considerable dilution of the plasma in the relevant vessels.

The veins of the filtering angle, which are supported by scleral strands, are, as far as the author is aware, the only minute vessels of the body which need not collapse as soon as the external pressure exceeds the internal. Consequently, in these vessels, the osmotic pull of the plasma colloids and the hydrostatic push can co-operate. This fact allows higher driving forces for fluid absorption than elsewhere and dilution of the streaming plasma by absorbed fluid should consequently more easily become evident.

Consider such a vessel. If it were completely filled with blood, the situation would be simple. The driving force behind the passage of aqueous humour would then be the sum of the hydrostatic pressure difference and the colloid-osmotic attraction of the blood plasma. However, the blood in the vessel is not pure: it becomes more or less diluted by aqueous humour when it absorbs the efflux from the anterior chamber. This reduces the osmotic attraction along the path of the blood through the vessel. When the intra-ocular pressure rises and more aqueous humour is forced out hydrostatically, this dilution will become greater and the colloid-osmotic attraction consequently smaller. When the intra-ocular pressure falls, the blood in the vessel becomes less diluted, and the colloid-osmotic attraction for aqueous humour rises. Plainly, we have here a mechanism that tends to keep the flow of aqueous humour constant, the direct opposite of an overflow mechanism. The degree of constancy achieved is clearly dependent on the rate of the blood stream through the vessel. When there is no blood supply at all, the colloid-osmotic factor disappears. The rate of efflux of the aqueous humour will then be in direct and simple proportion to the hydrostatic factor; there will be no stabilization of aqueous flow. When there is a very rapid flow of blood, the converse will be found—*i.e.*, a constant and fairly high osmotic attraction, which admittedly reduces the relative importance of the variable hydrostatic factor, but on the other hand does not stabilize the flow of aqueous absolutely. The stabilization tendency only appears in intermediate cases, but here the quantitative relations become so complicated that they cannot be treated in words. I have therefore made a mathematical calculation of how the fluid exchange over a vessel wall behaves, taking into account the change of the colloid-osmotic pressure which the plasma undergoes when it flows through the vessel.

I have not been able to find any previous treatment of this problem, which is one of general physiological interest. This may possibly be because the general case of the problem leads to a differential equation which cannot be solved exactly. The difficulty arises if one tries to pay regard both to the osmotic conditions varying along the

vessel, and to the variations in the hydrostatic lateral pressure along its course. As we are dealing with a venous vessel with relatively large surface and cross section a first approximation can ignore the differences in lateral pressure which may exist between different parts of the vessel: these can only be very small in relation to the other active-forces. We therefore assume that there is the same hydrostatic pressure in the whole vessel as far as this comes into contact with the aqueous humour.

Thus, we consider a linear vessel, through which blood flows. The lateral pressure in the vessel is everywhere equal to p_v . The colloid-osmotic pressure p_o is a function $p_o(l)$ of the position l . On entering the vessel $p_o = \bar{p}_o$. Outside the vessel there is aqueous humour under the intra-ocular pressure of p_i . (It is in relation to the aqueous humour that the colloids have the osmotic pressure of p_o — by this definition, we avoid most of the complications that might arise from considerations of Donnan effects). The blood streams into the vessel with the volume rate of V_i for plasma. During its passage through the vessel it takes up a certain volume of aqueous humour, partly by osmotic attraction and partly by hydrostatic filtration. The volume absorbed per unit of length and time, the specific absorption rate, will be called a . It is a function of l . The quantity absorbed in the vessel up to a point l is called A . We then have

$$A = \int_0^l a dl \text{ and } a = \frac{dA}{dl}$$

At the point l , the plasma has already been diluted to a certain degree. If there has been complete mixture, the degree of dilution is $V_i/(V_i + A)$. The diluted plasma has a lower colloid-osmotic pressure. We will assume for the sake of simplicity that the pressure is proportional to the concentration. This is not strictly so, and we shall be discussing what the simplification implies. On this assumption, however, the colloid-osmotic pressure at the point l will be

$$p_o = \bar{p}_o \cdot \frac{V_i}{V_i + A}$$

The hydrostatic pressure head is everywhere $p_i - p_v$.

Let us express the hydrostatic driving force in terms of the colloid-osmotic pressure of the undiluted plasma, \bar{p}_o , and define a relative hydrostatic pressure γ as

$$\gamma = \frac{p_i - p_v}{\bar{p}_o}$$

In the normal eye, γ cannot be much larger than about 1.0 or much less than 0.

The specific absorption rate $\alpha \equiv \frac{dA}{dl}$ is the product of the driving force and the specific filtration capacity of the vascular wall, g , at the point l :

$$\alpha = \frac{dA}{dl} = g \left(\bar{p}_o \gamma + \bar{p}_o \cdot \frac{V_i}{V_i + A} \right)$$

The integration of this differential equation using the boundary conditions ($l=0$, $A=0$), gives

$$\bar{p}_o \int_0^l g dl = \frac{1}{\gamma^2} \left[\gamma A - V_i \ln \left(1 + \frac{A}{V_i} \cdot \frac{\gamma}{1 + \gamma} \right) \right]$$

On the left we have the absorption rate for aqueous humour which the vessel would have had for purely osmotic reasons if it had been filled with pure plasma. We will call it O . We express the true absorption A and the plasma flow V_i in O as unit.

$$A = \alpha \cdot O; V_i = v_i \cdot O$$

Insertion of this and rearrangement finally gives the relation sought, even if only implicitly, between relative absorption rate, α , and relative hydrostatic pressure, γ . As parameter we have v_i , the relative plasma flow:

$$\alpha = \gamma + \frac{v_i}{\gamma} \cdot \ln \left(1 + \frac{\alpha}{v_i} \cdot \frac{\gamma}{1 + \gamma} \right)$$

For $\gamma=0$ this expression is indefinite. The limiting value, however, is:

$$\alpha_c = \sqrt{v_i^2 + 2v_i} - v_i$$

The equation cannot be evaluated exactly for other values of γ . Fig. 1 shows a number of solutions for different parameter values, obtained by graphic and numerical approximation. This has been taken to a point where the errors of the single ordinates do not exceed some few per cent.

The result of taking plasma dilution into consideration is visible in two properties of the graphs of Fig. 1.

First, the whole level of the curves, inclusive of the intersection with the ordinate axis, is dependent on the rate of plasma flow, v_i . Without plasma dilution, they would all start at $\alpha=1.0$ for $\gamma=0$. Secondly, the slope of the curves is smaller than 1.0 and especially small in certain pressure regions, where an inflection occurs. If there were no plasma dilution, the slope would have been 1.0 everywhere. This now only is the case for $v_i=0$ and ∞ .

The smallest slope is found at the initial part of the curve for the smallest plasma flow, $v_i=0.01$. Here, consequently, the highest degree of absolute stabilisation of aqueous flow by the "dilution mechanism" occurs. The slope is only 0.7 and is even less for

negative γ and smaller v . With higher rates of plasma flow, the corresponding region, which is visible in the higher curves as a small inflection, is displaced towards higher γ values and become less and less important.

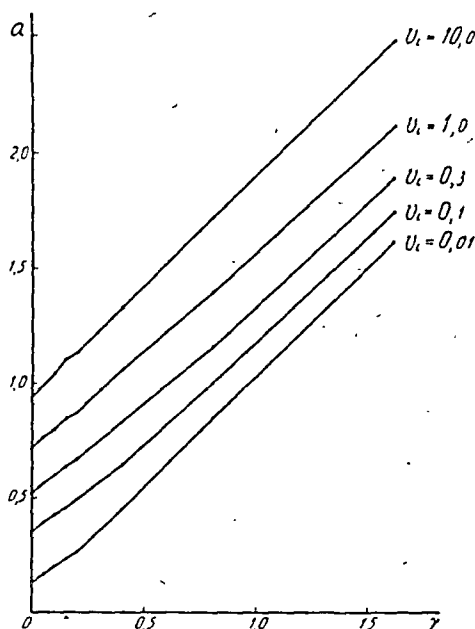


FIG. 1.

The rate of fluid absorption by a vessel under combined osmotic and hydrostatic influence.

Abscissae: γ = relative hydrostatic pressure head;

Ordinatae: a = relative rate of fluid absorption.

Parameter: v , = relative rate of plasma flow through the vessel.

For $\gamma > 0$, the osmotic and hydrostatic forces co-operate, for $\gamma < 0$ they oppose each other. All the curves intersect at $a = 0$, $\gamma = -1.0$. Consequently the slope gradually decreases towards the left. The curve for $v = 0.001$ is steeper than that for $v = 0.01$ in the interval shown.

In Fig. 1 the reduction of slope caused by the "dilution effect" appears not to be very important except in restricted regions and at low rates of plasma flow. However, in the derivation of our formula we have neglected certain factors which certainly will increase the "dilution effect" considerably.

First of all, we have assumed that the colloid-osmotic pressure sinks in proportion to the degree of dilution of plasma. As a matter of fact, it sinks considerably more (see Meyer 1932 for references and discussion). This naturally increases the dilution effect.

Furthermore, we have assumed that complete mixing between plasma and aqueous takes place in the vessel. This is not quite impossible, but on the other hand it is very probable that a certain

degree of stratification occurs at higher rates of aqueous flow, so that the fluid actually in contact with the vessel's wall is more dilute than the bulk of the fluid. This would be in agreement with the observations of Ascher and Goldman on man, where stratification of the blood and aqueous in episcleral vessels has been directly observed. A mechanism of this kind would materially increase the "dilution effect."

Finally and this is probably the most important point, we have treated only one vessel. Let us consider the situation if there are several vessels connected "in parallel" in the electrical sense, at varying depth in the sclera. A considerable influx of aqueous into those who are most accessible will deflect the blood stream into other vessels which have less contact with the aqueous. The plasma in the most accessible vessels will then be even more diluted than in our calculations, with a corresponding reduction in osmotic pull and a tendency towards absolute stabilisation of the rate of aqueous flow.

Until now, we have discussed the matter with regard to the absolute amount of aqueous flow and the stabilisation of this absolute amount. With respect to the regulation of the intra-ocular pressure, however, the relative variations of aqueous flow with changing intra-ocular pressure are more important. Summarizing the situation from this point of view we find the following:

(a) If the plasma flow through the trabecular veins is considerable, there is possibly not much absolute stabilisation of aqueous flow but instead a considerable "basal" flow even at zero hydrostatic pressure head. This results in a considerable relative stability of aqueous flow in spite of changing intra-ocular pressure.

(b) If the plasma flow through the trabecular veins is small, there probably is only a small "basal" flow at zero pressure head but instead a considerable absolute stabilization of aqueous flow. This also results in a considerable relative stability of aqueous flow in spite of changing intra-ocular pressure.

Thus we find that the existence of an osmotic factor in the aqueous outflow mechanism reduces the efficiency of the drainage mechanism in its rôle as a stabilizer of intra-ocular pressure in several ways. The osmotic factor both absolutely and relatively reduces the rate of change of aqueous flow with pressure, it tends to keep up a constant flow of aqueous.

The theory given thus explains the experimental findings presented in the previous section at least qualitatively. Moreover, it is in excellent agreement with some findings by Friedenwald and Pierce (1932). These authors observed, that if the rate of inflow of saline into a dog's eye is plotted as a function of pressure, the curve shows a definite inflection where the increase in flow per unit pressure increase is much smaller than at higher and lower pressures. Some of their results are reproduced in fig. 2. There can hardly be any

doubt that this behaviour of the curve is due to the plasma dilution mechanism discussed above.

In the experiments of Friedenwald and Pierce, the normal intra-ocular pressure is stated to have been 3-10 mm. Hg below the beginning of the inflected region. A pressure drop of this magnitude could easily be caused by the general anaesthesia. Moreover, the intra-scleral venous pressure could have been higher during anaesthesia. It seems not impossible, therefore, that in the unanaesthetized

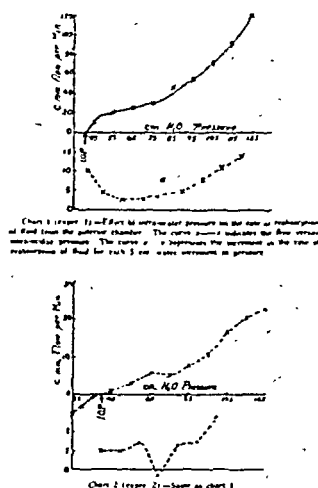


FIG. 2.

The rate of inflow of saline into the eyes of anaesthetized dogs. From Friedenwald and Pierce (1932).

dogs, the intra-ocular pressure was high enough to allow the drainage mechanism to operate in the inflected region, where aqueous flow is stabilized. The results obtained above for the normal rabbit's eye may perhaps indicate that this holds for the rabbit too.

How the situation is in man remains to be seen. The results of this study are not directly applicable to man, who has no trabecular veins. Friedenwald (1936) has made it seem very probable that the aqueous in the canal of Schlemm is mixed with a certain amount of plasma derived from special arterial connections. On principle, therefore, a similar kind of dilution mechanism could very well operate in the canal of Schlemm too, keeping the flow of aqueous more or less constant and decreasing the pressure-stabilizing properties of the drainage mechanism.

IV.—Concluding remarks

We have now arrived at the end of this investigation. Starting with the expectation of finding properties of the aqueous drainage mechanism which might be useful in stabilizing the intra-ocular

pressure, we have arrived at quite the contrary result. Experimental as well as theoretical findings point to the existence of a mechanism which, far from being suitable for stabilizing the intra-ocular pressure, is suitable for stabilizing the rate of flow of aqueous instead. What kind of physiological function could such a mechanism fulfil?

The answer lies near at hand if we once stop to consider the aqueous as merely something that causes intra-ocular pressure. The aqueous is the blood of the lens. What we have stumbled upon is a mechanism by means of which a fairly constant circulation of aqueous could be maintained in spite of the changing intra-ocular pressure.

In the "architecture of bodily function," a hierarchy of regulations exists. We have a regulation of blood pressure by the baroreceptors, but this regulation is subordinated to the need for adequate blood flow through vital and working organs. In case of conflicting purposes, the blood pressure regulation just gives in.

This simile may be extended to the eye. There is possibly a regulation of the intra-ocular pressure to which the drainage mechanism could possibly contribute. But this regulation seems to be subordinated to the need for a constant aqueous flow, which gives the lens a constant environment. In the cataracts which accompany various disturbances of intra-ocular pressure and aqueous flow we see what may perhaps be the result of overtaxing this mechanism. In fact, it is tempting to go even further: might not the glaucomatous pressure rise be considered, at least in some cases, as an adaptation, securing an adequate circulation of aqueous in face of impaired drainage facilities?

Summary

1. A new method of studying the dependence of aqueous outflow on intra-ocular pressure in the completely intact eye is described and applied to the rabbit. It is based on determinations in the aqueous of equilibrium concentration and rate of entrance of a substance at different levels of intra-ocular pressure.

2. Unilateral carotid occlusion does not change the equilibrium concentration of radiosodium. This shows that the change in aqueous outflow caused by the operation and the concomitant eye pressure drop is proportionally equal to the change in rate of entrance of radiosodium into the aqueous humour.

3. Unilateral carotid occlusion reduces the rate of entrance of radiosodium into the aqueous humour by only about 7 per cent., probably somewhat less. This shows that the reduction in aqueous outflow is percentually smaller than the reduction in intra-ocular pressure and hydrostatic pressure head. Thus, there must be some kind of relative stabilization of the rate of aqueous flow.

4. The influence of the hydrostatic driving force would be less if osmotic forces participated in the aqueous drainage mechanism.

Mathematical treatment of fluid absorption by a vessel under combined osmotic and hydrostatic action shows that if the diluting effect of the absorbed fluid is not insignificant, a certain absolute as well as relative stability of fluid absorption will obtain, in spite of changes in the hydrostatic pressure relations.

5. The results indicate the existence of an aqueous drainage mechanism which tends to keep the rate of aqueous flow constant even at the expense of the stability of the intra-ocular pressure. Such a mechanism could be significant from the point of view of the lens.

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A CASE OF BILATERAL GENUINE IRIS-ATROPHY*†

BY

IDA CZUKRÁSZ, M.D.

A lady, aged 24 years, came to the clinic complaining of intolerable headache and bad vision of the right eye. The left eye was always blind. Clinical findings: sight of right eye finger counting 1 metre,—8.0 D = 1/3; left, no light perception. Pressure on both sides 56 Hg mm. Right eye: eccentric pupil directed to 3 o'clock, roughly triangular in shape reaching almost to the iris-root. Pigment border nasally below and above is intact. Iris tissue very atrophic, the iris-frill can be seen only above and below. Temporally

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† Report from the Univ. Eye Clinic Debrecen. Leader: Prof. Kettesy.

there is a big defect from 8-11 o'clock extending to the angle of the anterior chamber. Between the hole and the pupil a 5 mm. iris strip is left with lack of iris-frill and pigment border. The defect is bridged over by two fibres of iris tissue. Some part of the posterior pigment layer of the iris is adherent to the lens-capsule. The lens is transparent, vitreous normal and so is the cornea. On the fundus there is a venous pulse and deep glaucomatous excavation. Visual field on the right side contracted, only a quadrant remains in the upper internal area (Fig. 1). Left eye: corneal surface slightly stippled. The pupil is continued to 12 o'clock in a congenital coloboma but there is no defect in the ciliary body nor in the choroid. Iris tissue atrophic, pigment border continuous with the angle of the anterior chamber but reduced, the iris-frill is well shown. Two

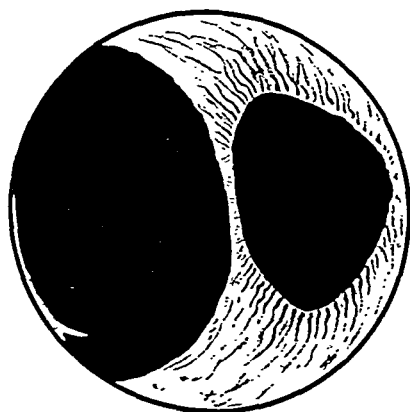


FIG. 1.



FIG. 2.

holes can be seen in the region of the iris-root one at 11 o'clock, the other from 1-4 o'clock that includes all the layers of the iris. The greater defect is divided in two by a stout bundle of trabeculae. There is a slight iridodonesis, the lens and the vitreous are normal and in the atrophic disc is a deep glaucomatous excavation (Fig. 2).

Regarding these findings, we could find neither macroscopic, nor slit-lamp evidence of previous inflammation in the left iris—an atypical coloboma is seen. We made the diagnosis of genuine iris-atrophy.

The genuine or essential iris atrophy is a well known but rare disease. In the University Eye Clinic, Debrecen, we have seen only two cases during the last 25 years. The first was published by Prof. Kettesy (Kreiker) in 1923. He observed the alteration in a lady aged 41 years, the other patient was a man aged 40 years, in whom there was a bilateral iris atrophy which progressed to total aniridia.

Regarding the genesis we were trying to find some solution. Clinical and laboratory examinations were made for tuberculosis, lues, endocrine aberrations without any positive result.

Reports in the literature show in some cases that hyper-sensibility of the thyroid and suprarenal glands could be proved, but this had no importance in the prognosis and absolute glaucoma.

As far as we can see, an explanation of this rare disease can be given only by the embryological dynamics, by that regressive power which is able to set in motion the pupillary evolution in the fourth month of foetal life. The process finishes soon enough, sometimes even before the pupil is completed. When this happens a persistent pupillary membrane is the result. Exceptionally a trauma or some other influence can regress the pupillary membrane during life also. Kreiker's published case is a nice example, and Wolfrum's experiments seem to be able to prove this assumption too.

We come to the conclusion that the regressive function of the iris is influenced by an inhibitory factor. One can imagine, if in these embryological mechanics, in the activity of action and reaction, the faintest disturbance arises, the balance overturns, and develops, what Riger called, the dysgenesis mesodermalis.

No doubt, the solution of the question will be given by getting more insight into the embryological and respectively biological happenings.

From the practical point of view, the treatment of the secondary glaucoma seems to be the most important. Ciotola, Licskó, de la Vega, Post, Barkan and others have done different anti-glaucomatous operations. Trephining and irido-sclerectomy are the most frequent operations. Csillag and I. Grósz do cyclodialysis with good results.

If we take the figures above, which show the huge defects in the iris the decision of cyclodialysis is obvious. There

is only one question, where to make the operation, which part of the ciliary body shall we detach? We decided, referring to the intact part of the iris, at the insertion of the inferior rectus muscles. Next day the pressure was subnormal (12 Hg mm.) and for half a year there has been no alteration in the pressure.

As far as we are concerned we would propose in cases of genuine iris-atrophy combined with high pressure a cyclodialysis a tergo (secundum Blaskovics) at the insertion of inferior rectus muscle.

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THE DEVELOPMENT OF MEDICAL STUDIES IN BRITAIN: OPHTHALMOLOGY*

BY

R. R. JAMES

Earliest Times

ENGLISH ophthalmological history may be said to start with the Roman occupation of Britain. From the large number of oculists' stamps which have been unearthed on Roman sites we know that a good deal of local treatment of eye conditions by way of collyria and ointments was practised at this early date. After the withdrawal of the legions there is a long gap in our knowledge; in fact nothing is known until we come to the Anglo-Saxon *Leech* [medical] books and Herbals. Here again, treatment was mainly by local applications of infusions of herbs and the secretions of animals, such as gall mixed with honey, and even of human urine. Charms also played a large part and we may say that Anglo-Saxon ophthalmology has every appearance of having been largely futile.

The Norman Conquest and after

The Norman Conquest did little to improve the practice of ophthalmology but the 13th century saw the beginnings of optics

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in Britain. Grosseteste, Bishop of Lincoln, was the teacher of Roger Bacon (died 1294), who proved that spherical *plus* spheres would be of use for reading in old people; while John of Peckham (died 1294), later Archbishop of Canterbury, is credited with the discovery of the use of concave refracting surfaces, and his *Perspectiva Communis* was for the next few centuries the only textbook on optics to be used in England.

In the year 1377 John of Arderne wrote his little book entitled *De cura oculorum*, of which manuscripts in Latin and in 15th-century English exist among the *Sloane Manuscripts* at the British Museum, and in the Library of Emmanuel College, Cambridge. This booklet is a mere compilation of other people's views, much of it being taken from Lanfranc. English ophthalmology had, in fact, made very little progress since Anglo-Saxon times.

The Sixteenth and Seventeenth Centuries

The closing years of the 16th century saw the publication of two small books dealing with ophthalmology. One was Walter Bayley's *Briefve Treatise Touching the Preservation of the Eye-sight*, and the other a translation (probably by A. Hunton, of Newark-on-Trent) of Guillemeau's *Maladies de l'Œil*.

Early in the 17th century Richard Banister, of Stamford, brought out a duodecimo of 478 pages containing five separate treatises. Of these the first 112 pages are Banister's own contribution, and this section is named Banister's Breviary. Though he was an itinerant oculist it is obvious that Banister was a much more honest practitioner than the rest of his ophthalmic contemporaries. He it was who first pointed out the cardinal sign of hardness of the eyeball in cases of *gutta serena*, in this case glaucoma. The significance of his observation was not recognised, and raised intra-ocular tension was forgotten for the next 150 years. Banister was also almost certainly the author of the manuscript in the British Museum (*Sloane Manuscript*, 3801) which throws much light on the manners and customs of the itinerant charlatans of this date. Several of these are named, such as Luke of Erith, Mr. Surphlete of King's Lynn, and Henry Blackborne. The last-named is severely criticised by Banister, and it is a sign of the looseness of the times that Blackborne in 1605 received the Archbishop of Canterbury's licence to practise in diseases of the eyes. Richard Banister must have been a remarkable man and has deserved well of ophthalmology.

Turberville of Salisbury (died 1696) was a qualified medical man in an age of unqualified quacks. He had a large practice and made a valuable contribution to ophthalmology in extracting an iron particle from the cornea with a magnet. Another qualified English physician of this time was William Briggs, who published the

Ophthalmographia and the *Nova Visionis Theoria*. He described the papilla of the optic nerve in 1676 and, in 1684, recorded in the *Philosophical Transactions of the Royal Society* a case of night-blindness. Dimness of vision following the administration of Peruvian bark in fever was described in 1681 by a general practitioner at King's Lynn in a letter to Briggs.

The Eighteenth Century: Charlatinism and the Foundations of Modern Ophthalmology

The 18th century was the age of ophthalmic quackery, but it saw the foundations of modern ophthalmology laid by such masters as Cheselden, Sharp and Warner. Cheselden devised the operation of iridotomy in the making of an artificial pupil, and Sharp and Warner perfected the operation of extraction of cataract, a great advance on the age-long method of couching [displacement of the cataract from the visual axis].

Duddell, a pupil of Woolhouse, wrote a good account of diseases of the cornea in 1729. Later in the century, James Ware wrote on syphilis in connexion with ophthalmia, and brought some order out of chaos in the matter of conjunctival diseases. Gataker wrote on the anatomy of the eye and on the use of belladonna, and Wells on double vision.

The chief 18th-century ophthalmic quacks were William Read, Roger Grant, and the Chevalier Taylor. Read and Grant were illiterate; the former was knighted by Queen Anne. The Chevalier Taylor was quite a remarkable person. In professional knowledge he was often far ahead of his time, but he practised all the arts of unblushing effrontery and charlatanism. He was oculist to George II, and his son and grandson followed in his footsteps though they were not of the same calibre. Eighteenth-century Royalty was singularly unfortunate in its oculists, as George Coats pointed out.

The end of the century, in 1794, saw the publication by John Dalton of the history of his colour blindness. Thus, it will be seen that up to the end of the 18th century ophthalmology had not advanced very far, but better times were to come.

Rational Ophthalmology displaces Charlatanism

The year 1805 saw the foundation of *Moorfields Eye Hospital by J. C. Saunders, and this more than anything else struck the death blow to the quackery of the previous century. Provincial Eye Hospitals were founded at Exeter in 1808, Bristol in 1810, and Manchester in 1814. The Royal Westminster Ophthalmic Hospital was founded by Guthrie in 1816, the Central London

* Now the Royal London Ophthalmic Hospital.

Ophthalmic Hospital in 1843, the Western Ophthalmic Hospital in 1856 and the Royal Eye Hospital in the year following. The first course of lectures on diseases of the eye was given by Guthrie at the Royal Westminster Ophthalmic Hospital.

An earlier epoch-making date in ophthalmological history is 1801, when Thomas Young published his paper on the mechanism of the human eye in the *Philosophical Transactions*. He described astigmatism and measured the amount of the astigmatism in his own eye. His table of optical constants has been only very slightly modified by modern research. His theory of colour vision postulated the presence in the retina of three "fibres," which correspond to the colours red, green and violet respectively. This theory was later resuscitated by Helmholtz and is known as the Young-Helmholtz theory. Young's experiments on interference strongly supported the undulatory theory of light already adumbrated by Sir Isaac Newton and Huygens.

Ophthalmological Literature and Societies

The first real text-books of ophthalmology in Britain belong to the first half of this century. In 1830 William Mackenzie of Glasgow brought out his great work on diseases of the eye. It was far ahead of any previous text-book on the subject and it ran to a fourth edition. Mackenzie was a master clinician, and was the first surgeon to give an adequate account of sympathetic ophthalmitis. Sir William Lawrence's text-book appeared in 1833, and Richard Middlemore's in 1835. Before this, Travers had brought out his synopsis of diseases of the eye in 1820. It ran to a third edition. J. C. Saunders published a book before his untimely death, and did much to revolutionise the treatment of congenital cataract by insisting on early discission. In this he followed the practice of Woolhouse, an English surgeon, who was resident in Paris for many years in the previous century.

Wardrop's *Essay on the Morbid Anatomy of the Human Eye* laid the foundations of ophthalmic pathology; Tyrrell's *Diseases of the Eye*, in two volumes, appeared in 1840; and Dalrymple's splendid atlas belongs to about this date.

The year 1881 saw the foundation of the *Ophthalmological Society of the United Kingdom* with Sir William Bowman as first president. It grew out of informal discussions in the house-surgeon's room at old Moorfields, and of the Committee appointed to make arrangements for its foundation Sir Thomas Barlow was at the time of his death the last survivor. Sir H. Lindo Ferguson of New Zealand is also still with us as an original member of the Society. The Society now has, as affiliated members, the *Oxford Congress*, founded by R. W. Doyne, the *North of England*

Ophthalmological Society, founded by Percival Hay and J. Gray Clegg, the *Midland and South Western Societies*, the *Irish Ophthalmological Society*, and the *Scottish Ophthalmic Club*. A volume of *Transactions* has been published each year without a break since 1881, and the series forms a rich mine of ophthalmological facts.

In 1857 the staff at Moorfields began the publication of the invaluable *Ophthalmic Hospital Reports*, which ran to 20 volumes. In 1864 J. Z. Laurence and T. Windsor began the publication of the old *Ophthalmic Review*. It came to an untimely end 3½ years later and the new *Review* was started in 1881 by Priestley Smith of Birmingham and Karl Grossmann of Manchester. The *Ophthalmoscope* was founded by Sydney Stephenson in 1903. In 1917 the first number of the *British Journal of Ophthalmology* appeared. It incorporates the *Moorfields' Reports*, the *Ophthalmic Review* and the *Ophthalmoscope*, and has continued without a break to the present day.

Ophthalmic Instruments and Operations

English ophthalmology has been responsible for many inventions of ophthalmic instruments. It should not be forgotten that William Porterfield (died 1771) devised the first optometer. Charles Babbage, in 1848, constructed an ophthalmoscope which left little to be desired. He showed it to Wharton Jones, who, alas! did not realize the importance of the means of research thus placed at his disposal. Babbage was a mathematician, not an ophthalmic surgeon, and finding that Wharton Jones was not interested in his model he took no further steps and it was left to Helmholtz to bring out his instrument in 1851. Tyrrell devised the iris hook which bears his name; Bowman a trephine for the eye-ball; Mules of Manchester first suggested the insertion of a glass globe in the sclerotic after evisceration of the eyeball, and his practice was later modified by Frost and Lang, who inserted globes of glass or metal into the orbit.

Corneo-sclerotic trephining for chronic glaucoma was first proposed by Freeland Fergus of Glasgow in 1909. In the next year Elliot, of Madras, improved the operation by splitting the superficial layers of the cornea in order to make more sure of tapping the anterior chamber, and the operation has been known by his name ever since. Herbert, of the Indian Medical Service, also made important additions to our anti-glaucoma methods at this date.

Stanford Morton's ophthalmoscope was for years the best of its kind, though Frost's instrument was a close second. Frost also brought out an extremely effective model eye for teaching purposes.

Ophthalmology as a Speciality

In the early years of the last century ophthalmology was still, in Britain, a part of General Surgery; Sir William Bowman was the first general surgeon to give up his surgical work at King's College Hospital in order to become a pure ophthalmic specialist. With George Critchett, Bowman was responsible for the foundation of this speciality. But Critchett, and even Jonathan Hutchinson, were general surgeons primarily and ophthalmic surgeons in the second place.

Bowman's name is a household word in ophthalmology. Bowman's membrane, his probes, and "stop" needles are known everywhere; but it was his guiding hand which placed ophthalmology as a speciality on a sure footing. Lawrence wrote on syphilis of the eye. Hutchinson's monograph on syphilitic disease of the eye and ear is a classic, and he was the first to describe the notched incisors in congenital syphilis which will always be known as "Hutchinson's teeth." It is reputed that a French surgeon who visited Moorfields in the early 60's rushed into the out-patient room exclaiming "Where is Monsieur Hutchinson? I want to see his teeth."

Hutchinson's main assistants were Waren Tay and Edward Nettleship. Tay never cared for publicity and was glad to remain in the background. Hutchinson's mantle may be said to have fallen on Nettleship, who had a profound influence in the teaching of ophthalmology in his generation.

Argyll Robertson first described the tabetic pupillary reactions which have ever since borne his name; and in an earlier decade Arthur Jacob, of Dublin, first described the layer of rods and cones in the retina in 1819.

One of the first atlases of the fundus was that of Liebreich who was ophthalmic surgeon to St. Thomas's Hospital. He had been turned out of Paris at the beginning of the Franco-Prussian War of 1870, and came to England. There have been several good atlases since his day, notably that of Frost, the pictures in which are still unsurpassed.

Ophthalmic pathology has been well served by the long line of curators of the museum at Moorfields Hospital. Nettleship, Lawford, Treacher Collins, all made notable contributions in this subject, but George Coats, who died in 1915, probably contributed more papers of lasting value than any other, with the exception of Sir John Parsons whose magnificent *Pathology of the Eye* in four volumes has long been a credit to British ophthalmology.

For many years the importance of the state of the refraction of the eye was overlooked and underestimated. A case of Brudenell Carter's published in the Clinical Society's *Transactions*, in 1875,

was one of the first to call attention to the importance of this branch of work; and partly in consequence a demand arose for hand-books on refraction; among these, that of Hartridge ran through many editions and was deservedly popular.

In the field of medical ophthalmology, the manual by Sir William Gowers was pre-eminent for many years: its third edition was brought out by Marcus Gunn who, from his experience as Ophthalmic Surgeon to the National Hospital, Queen Square, did much to advance ophthalmology in its relations to neurology. Perimeters have been invented by McHardy, by Priestley Smith and by Sir William Lister. Priestley Smith won the Jacksonian prize at the Royal College of Surgeons in 1878 with his essay on glaucoma. Few men have had more influence on their speciality of recent years than this celebrated Birmingham ophthalmologist.

Ophthalmological Education

Postgraduate education in ophthalmology is given at the universities and at all the great eye hospitals in London and the provinces. Courses of lectures which include all ancillary subjects such as optics, operative surgery, bacteriology and pathology are given during the terms, and at the same time the student takes his place as clinical assistant to one or more of the members of the senior staff in the out-patient department. If time is no objection, he is wise to hold the post of House Surgeon for a year or more at one of the Eye Hospitals. Even if he does not, he obtains a thorough grounding in clinical ophthalmology, and it is this aspect of teaching which has produced so many of the great names of British ophthalmology during the past hundred years. Special diplomas notably the D.O. Oxon. (1907) and the D.O.M.S. (1920) are awarded to students who pass the qualifying examinations, and for many years ophthalmology has been one of a number of special subjects which can be taken in the examination of F.R.C.S. (Edinburgh).

Preventive and Social Aspects of Ophthalmology

Steady progress has been made during the present century both in prevention of blindness and in amelioration of the conditions of the blind. Blind pensions are now awarded, and ophthalmic monetary benefits under the National Health Insurance came into force in 1925.

Early in its history the *Ophthalmological Society* appointed a deputation, led by Bowman and Hutchinson, to interview the Home Secretary and draw attention to the ravages caused by *ophthalmia neonatorum*. It was pointed out that this affection is largely preventable, and it was suggested that the Registrar

of Births should hand each person registering a birth a printed paper telling him what to do if the baby's eyes showed any signs of inflammation. No action was taken and little was done by the authorities until the *Metropolitan Asylums Board* established St. Margaret's Hospital in 1918 for the treatment of *ophthalmia neonatorum*, affording facilities for the admission to hospital of both mother and child. The *Board* also did good pioneer work by establishing, in 1903, residential schools for the treatment of trachoma in children, at the same time ensuring the continuance of their education.

The *London County Council*, at the instigation of Mr. James Kerr and Mr. Bishop Harman, made a notable advance by segregating partially-sighted children in special—so-called “myope”—schools. Valuable reports on the prevention of blindness have emanated from Glasgow in 1926 and in 1942, and from the Union of Counties Association for the Blind in 1932; and the Ministry of Health has at last set up a Standing Committee on the Prevention of Blindness.

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ANNOTATIONS

On second opinions

A doctor, in discussing with a friend the foibles of patients in general, is reputed to have said that if he suggested a consultation, it was because he didn't know what was the matter, and that if he pooh-pooh'd the idea as unnecessary, he was afraid of showing his ignorance. As in a large number of cases it is impossible to be dogmatically sure of one's diagnosis it is a wise plan to have a second opinion; and in many cases a consultation will be of great help in settling the line of treatment to be adopted. An intelligent patient will understand the problem if it is placed before him squarely, and will be willing to pay the necessary fee. In the case of what Shakespeare somewhere calls “a blinking idiot” a second opinion is even more necessary in order to safeguard oneself.

We do not mean to imply that extra advice should be proposed in all cases. As a general rule a straightforward case of cataract ready for operation need not be referred to anyone else. Probably it would be wise in cases of diabetes with cataract to have a consultation, usually with a physician, before undertaking any operation at all.

A second opinion is often called for in cases of the more chronic forms of glaucoma. It should certainly be pressed upon a subject with a wounded eye in view of the possibility of the onset of sympathetic ophthalmitis. Border line cases between our own speciality and others, such as neurology and the nose and throat department should have this extra advice.

Sir William Bowman, in consultation, is said to have spoken shortly, in simple language and to have kept strictly to the point: and this attitude is recommended to those who have to act in this capacity.

The suspicious patient may assume that his doctor and the second opinion are acting in collusion against him, but this cannot be helped.

It is wise, if seeing a patient in his own home, to attend strictly to the business in hand when talking over the matter with the doctor who has called you in. Bearing on this point an apocryphal story is that a very plain looking lady arranged for her sister to be secreted in an annexe to the dining room so that she could hear and report what the doctors said to each other about the case. When the door was shut the consultant is reputed to have said "what a horribly ugly patient," to which the G.P. replied "my dear Sir, you should see her sister."

On appointment books and appointments

When the writer started in practice most surgeons relied on the appointment books supplied by Burroughs, Wellcome & Co. Their books were excellent, of handy size, well arranged and packed with useful information. They were supplied in two varieties, one for the consultant and the other for the general practitioner. Those who remember the book will recall that the publishers provided a specimen page to show how it should be used. Out-door appointments were to be entered in red ink, appointments at home in black. In our early days we often looked with envy on the specimen page. Appointments started at 9.30 a.m., and most of the spaces were filled up till tea time. The last entry. "Dinner, Med. Soc. Speech," always tickled our imagination. Only one hour was allotted to hospital visits, so we presumed that the hypothetical surgeon was not attached to any ophthalmic hospital. This page must have been prepared long before the days when each consultant had a secretary or a part share in one. When did the Victorian surgeon write his letters? Sir James Paget wrote most of his after dinner at night we are told in his life.

We have known elderly ladies being very angry at receiving a printed appointment card. One indeed, who was stone deaf, resented

receiving a typed note of her appointment signed by our secretary. One friend of our's said that he always wrote a personal note to every patient who wrote to him. This was on the advice of a very eminent Victorian surgeon, who had found that it paid to do this. Times have changed since the spacious days of the end of the last century, and nowadays nearly all appointments are made by telephone.

We always considered it a mistake to try and fit more work into the day than we felt we could honestly accomplish. Some patients very much resent being kept waiting. At times, when earlier patients have been delayed, this cannot be helped; but there is always a danger that if the surgeon be overtaxed as to time he may omit some part of the examination which could have been undertaken had there not been a number of other patients waiting to be seen.

It is a good plan to be at home for appointments only, and not to have people coming in in shoals in the expectation of being fitted in.

We remember a lady who was very angry at being kept 20 minutes for her appointment. She was so angry that she refused to accept our reasonable explanation, so at the end we suggested that she had better go away and see some one else. Her answer was—"I have already seen some one else and he kept me waiting even longer' than you have done. That is why I am so angry." She was gravely ill with high blood pressure and retinal changes, so we made excuses for her and did what we could.

BOOK NOTICES

Corneal Transplantation. By B. M. FILATOF. Odessa. 1945.

Filatof published in Odessa in 1945 a book, in the first part of which, he describes and discusses his operation for transplantation of the cornea, and in the second his stimulating tissue therapy. The indications for the transplantation of the cornea are the usual ones. But the surgical technique is different. Transplantation is indicated in cases of thick leucomata, covering the whole of the cornea; the tectonic one—on partially projecting staphyloma and fistulae; cosmetic one for amelioration of the leucoma, as a preparatory step to an optical one; a reconstructive one—first proposed by Elschnig; and lastly, a therapeutic one—to heal up parts of the cornea close to the graft—really a kind of Filatof's "stimulating tissue-therapy." The surgical technique is different for some forms.

Partial total transplantation (950 cases). The purpose is to form a transparent little window through the leucoma. A special frame

is applied in the eye and face of the patient and both lids are fixed to it by sutures—so that no pressure whatever is caused in the eye. Filatof uses retrobulbar anaesthesia, subconjunctival over the superior and inferior recti. He prepares a flap of the conjunctiva 11 mm. long and 8-10 mm. wide. After that he passes stitches through the flap and through the superior and inferior recti; he makes certain that the flap will cover the whole of the graft. After washing the donor's eye first with a solution of brilliant-green (1-500) he cuts out a disc from the centre of the eye by a special trephine and puts the trephine with the disc inside it under a glass cover. On the centre of the leucoma of the recipient a mark is made by brilliant-green with an instrument and the eye is fixed by stitches through the superior and inferior recti and by forceps, but without causing any pressure. Filatof uses a trephine, of which the important part is that its crown is first cylindrical (1 mm. from the cutting edge), the canal is hermetically closed, and so the air is compressed by the disc of the cornea and the aqueous after the anterior chamber is penetrated, and so the lens is always protected by the retained aqueous. After putting a drop of 1 per cent. atropine in the wound, Filatof puts the graft on the wound by pressing down the piston of the trephine. Then the flap of the conjunctiva covers the graft, epithelium towards the cornea, and the stitches are tied first the middle one and then the two lateral ones. Then Filatof removes the stitches from the superior and inferior recti muscles, then the lid fixation sutures. Subcutaneously he injects 1 c.c. of saline into the upper and lower lid to safeguard complete closure of the lids. The binocular dressing is changed daily. In six to seven days the conjunctival sutures are removed and the conjunctival flap excised. The same operation may be performed by another type of trephine with a metallic support, and a piece of ivory with two holes; and a metallic screw to fit the ivory piece to the support. The idea is to introduce the ivory piece before trephining a hole into the anterior chamber, so that when using the trephine the lens is protected and there is a safeguard against the prolapse of the vitreous. A lance-knife with the usual angle is used, but the cutting edge is only the triangular one, the straight one is not cutting. After the knife cuts the cornea, Filatof first introduces the ivory piece, and an assistant fixes the free end of it by a hook. After the excision of the opaque cornea, the whole instrument is removed, but the ivory piece is left inside. By moving it backwards the surgeon brings its larger hole against the wound, inspects the interior of the eye, removes pieces of the iris or the capsule of the lens and again moves the ivory piece into the initial position. The extraction of an opaque lens is left till later on. The graft, prepared as in the first operation, is put into the wound, the flap of conjunctiva is replaced, etc., as described above. If the operation is performed without the ivory piece, Filatof uses his

obturator. After trephining, the surgeon introduces an obturator, raises it so that the disc closes the wound, introduces the knife behind the obturator, then introduces the ivory piece, removes the obturator and proceeds as described above.

Post-operative complications are—keratitis and iridocyclitis and glaucoma (the latter 30 per cent.). Filatof uses as donors the living and the cadaver cornea. The cadaver eye is usually kept for a few days (1 to 3) at a temperature of 2-4° above zero; he does not use the eye of a person who has suffered from syphilis, or malignant neoplasms.

The operation for amelioration or partial (by layers) transplantation is done by Graefe's knife and by special knives of Filatof. The layers of the leucoma are cut off until iris or the pupil can be seen. Then the graft is cut from a cadaver's eye in a similar way and is put into the wound. The rest of the operation is as described above.

Cirurgia Ocular. By H. ARRUGA. Roy. 8vo., pp. XXXI, +888 with 1218 illustrations, 119 in colour. Salvat Editores S.A. Barcelona: Buenos Aires. 1946.

This is a veritable *magnum opus* of Dr. Arruga, who has returned to Spain and dates his preface from Barcelona.

There are two ways in which a text-book on operative surgery can be written: one is that in which the author describes his own procedures, giving little if any attention to alternative methods; the second is that which includes all those that custom has established as sound. The present work is of the latter kind though it is based for the most part on Arruga's own methods of practice.

The descriptions of the various operations are very full and the admirable illustrations fully portray each step. The surgery of the lens occupies 131 pages and that of retinal detachment 94. The latter chapter is superlatively good and the fine coloured illustrations, of which there are a great number, add greatly to its value.

Each section of the work includes a full bibliography, and that this is up-to-date is evidenced by the fact that several references to papers which have appeared in contemporary British and American journals of the war years are included. The book is well bound and well printed on art paper. When, as in this instance, the number of illustrations exceeds the number of pages no other kind of paper would have been suitable. It is a credit to its distinguished author, and to his publishers, and to Spanish ophthalmology in general, including Latin America.

British and American surgeons would we feel sure welcome an English translation of it and we hope that the author will see his way to providing one.

CORRESPONDENCE

MISTER or DOCTOR

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—Although strictly speaking the title of Doctor should be confined to those who *are* "Doctors," i.e. those who hold the degree of Doctor of a University, yet since this title is now so universally applied to any duly qualified medical practitioner I agree with the writer of the annotation on the above subject that it is questionable whether this "Mister - Doctor" distinction subserves any useful purpose now, although it may have done in the past in the days when the Surgeon was a sort of "plumber" called in by the Physician to do a piece of technical manual work.

As I see it the chief disadvantages of the use of the term "Mister" as applied to a Surgeon are:—

1. It is confusing to the lay public.
2. It is confusing to our medical colleagues in other countries.
3. It "covers" the "unqualified" medical practitioner, for how can the layman easily distinguish (if he wishes to do so) between two gentlemen with West End addresses in the Medical area, one being a reputable Surgeon medically qualified, the other being a not-medically-qualified "naturopath," both styling themselves "Mister"? Similarly how can the layman easily distinguish between a medically qualified ophthalmologist and an optical practitioner or ophthalmic optician, if both are just "Mister."

Now that we are on the eve of the establishment of a State Medical Service in which it is envisaged that sight testing opticians will take their official place, I would advocate that the medical men in the ophthalmic profession should for the sake of differentiation style themselves Doctor instead of Mister (just as they do in Scotland and also in Manchester).

I might add that I have no personal feelings on the subject and could not care less whether I am addressed as Mister, Doctor or just

Yours truly,

KEITH LYLE.

42, CHARLES STREET, W.1.
30th January, 1947.

MEPACRIN AND THE EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—Miss Ida Mann in her communication "Blue Haloes in Atebrin Workers" has solved the clinical appearance of what

was at one time a diagnostic problem among African troops in India. The self-inflicted ocular injury caused by putting powdered mepacrin in the conjunctival sac. Although it was by that time well recognised, the first case I saw was shown to me in the summer of 1945 by Major A. E. Wilson, Ophthalmic Specialist of 135 West African General Hospital. His very accurate description of this case is as follows. "The bulbar conjunctiva was stained in an area surrounding the limbus and extending laterally in an area corresponding with the palpebral fissure. The bulbar conjunctiva above and below were devoid of any staining and the lines of demarcation above and below were sharp. The palpebrae were clear apart from narrow bands at the posterior aspects of the lid margins. The cornea was hazy and had a yellow-green colouration as if fluorescein had been inserted into the conjunctival sac and had been incompletely washed out." Major Wilson had already made the diagnosis and having searched the soldier's kit had found a yellow powder which proved to be mepacrin. He later removed a portion of the affected conjunctiva and on chemical test it gave a positive test for mepacrin. The yellow staining in the cornea disappeared after, I think, some weeks. A number of these cases were seen both by Major Wilson and by Major Hollingsworth (40 West African General Hospital) among African troops. Both these specialists reported that there was a mild conjunctivitis in the early stages and that in some a striate keratitis was present. No haloes were complained of as far as I know.

One of the diagnostic difficulties in these cases was the fact that although the mepacrin powder was presumably put in the lower fornix the conjunctival staining, with its definite edges, was limited to the interpalpebral zone of the bulbar conjunctiva. Thus it appears that exposure, possibly associated with oxygen intake, may play an essential part in the ability of the cells to take up the staining granules. The narrow yellow line on the conjunctiva of the lower lid margin was very noticeable and not easily explained either. The "striate keratitis" noticed in some cases is no doubt the same as the corneal oedema produced by massive local application in Miss Mann's experiments.

As a self-inflicted injury this procedure was successful for in a country with a high incidence of jaundice some delay was inevitable before the diagnosis could be established, thus affording the soldier the change and rest that he desired.

Yours truly,

L. B. SOMERVILLE-LARGE.

2, FITZWILLIAM PLACE,

DUBLIN.

January 28, 1947.

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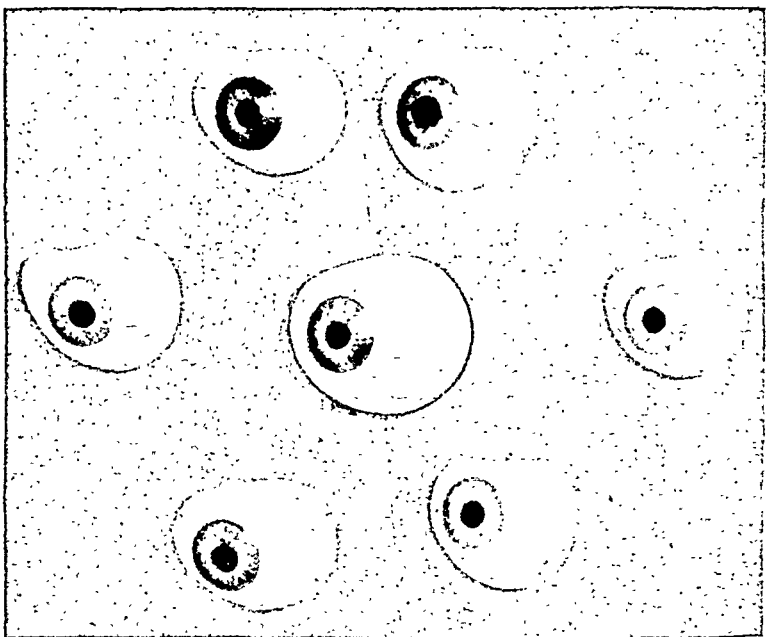
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24-27, THAYER STREET, W.1



ARTIFICIAL EYES IN PLASTIC

Artificial Eyes until the beginning of the war were invariably made of glass, and their production was entirely in the hands of a few very skilled craftsmen in this Country and on the Continent. Up to August, 1939, Theodore Hamblin, Ltd., employed Mr. Paul Asprion, of Vienna, at 15, Wigmore Street, and at their various provincial branches, where he made artificial eyes in glass while the patient waited. With the outbreak of war, these visits had to cease and steps were taken to develop the manufacture of artificial eyes in plastic material. The many difficulties of producing eyes in this material have been overcome, and they are now made throughout in plastic, no paper or glass being incorporated.



Eyes made in plastic have many advantages over those made in glass. They are life-like in appearance, comfortable in wear, are not affected by the secretions of the orbit, and above all, they are unbreakable.

Difficult shapes necessitated by war injuries, burns, etc., or thin shells to fit over shrunken or deformed globes, almost impossible to produce in glass, are quite possible in plastic.

Patients may be sent in to 15, Wigmore Street or to most of the provincial branches, where stocks of ready-made eyes are available from which selections may be made and fitted. Specially made eyes for more difficult orbits can be made with little delay. For these a carefully made mould of the orbit is first taken, and a special iris is produced in plastic. In such cases a second visit after the eye has been made is necessary for fitting.

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THE BRITISH JOURNAL OF OPHTHALMOLOGY

APRIL, 1947

COMMUNICATIONS

EXPERIMENTAL OBSERVATIONS ON THE INTRA-VITREOUS USE OF PENICILLIN AND OTHER DRUGS*

BY

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I. C. MICHAELSON and J. M. ROBSON

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Introduction

It is a matter of common experience that infection of the vitreous body is a major catastrophe which results almost inevitably in destruction of the eye. Interest, therefore, has been directed to new therapeutic agents which have been shown to be of value in the treatment of sepsis elsewhere in the body; in the hope that severe intra-ocular infection may also be controlled by them. Unfortunately, while drugs administered either orally or by injection readily reach most tissues in adequate concentrations, the vitreous is exceptional in this respect, in that drugs, present in the blood stream, do not easily diffuse into it. Hence, it is very difficult to produce adequate

* Received for publication, December 9, 1946.

concentration of therapeutic agents in the vitreous by their systemic administration.

It seemed desirable, therefore, to investigate the practicability of injecting these drugs directly into the vitreous, since it is known that they can produce their effect when applied locally to a site of infection. The experiments recorded below were made to this end and may conveniently be described under three headings. First, the effects of the drugs on the normal eye, secondly, diffusion of the drugs within the eye, and lastly, the value of certain drugs in the treatment of experimentally produced infections of the vitreous body.

METHODS

All experiments were performed on rabbits. Under ether anaesthesia the drugs were injected into the vitreous by means of needle puncture of the sclera in a region well behind the ciliary body, the needle being directed backwards to avoid possible injury to the lens. The needle used had a short bevell point, was 7.8 mm. long and the amount of solution (0.05 or 0.1 c.c.) delivered by the syringe was accurately controlled by means of a micrometer gauge (see Fig. 1.).



FIG. 1.

In most experiments a single injection was given, in a few the injection was repeated after 48 hours. Thereafter, the eyes were observed over periods of 10 to 128 days by the usual methods, particular attention being paid to the fundi and media. Finally, the eyes were removed and submitted to histological examination. The following drugs were used:—

1. Penicillin: Both commercial preparations (300-700 units/mg) and the pure sodium salt (1600 units/mg). The dose introduced was 2000 or 5000 units.

2. Sodium sulphacetamide: A 30 per cent. solution in pyrogen free water.

3. Marfanil: A 30 per cent. solution of Marfanil hydrochloride neutralised to pH 7.0.

4. V.335: (p-methyl sulphonyl benzylamine)—see Evans, Fuller and Walker (1944)—a 30 per cent. solution of the hydrochloride neutralised to pH 7.0.

Control experiments with normal saline were carried out.

Section 1. The effects of the Drugs on the Eye

METHODS

The effects of the drugs were observed on a total of 52 eyes of which 36 were examined histologically. Eight normal eyes, and 6 injected with saline were also examined histologically. Most of the material was fixed in 10 per cent. formol saline; that selected for examination by Nissl's method in 90 per cent. alcohol. While still in the fixative, the globes were divided equatorially and the posterior halves examined with a binocular microscope and the slit-lamp, and areas for section were selected. By this means, lesions seen and sketched in the living animal were identified and the correlation between clinical and histological findings ensured.

The tissues were embedded in paraffin, excepting those to be stained for fat, which were embedded in gelatin. All tissues were stained with haemalum and eosin; a few were in addition stained with scarlet red, with Nissl's stain, and with Marchi's method.

RESULTS

Great differences exist in the appearances of the fundi in apparently normal eyes of rabbits. While the usual appearance in pigmented animals is a fairly even granular one, not uncommonly pigment patches or clumps are seen, similar to those found in an old choroiditis in man. Histologically, however, such eyes do not show retinal abnormalities of the type to be described as resulting from the intra-vitreous injection of certain drugs. The vitreous opacities resulting from the injection of normal saline were slight and as a rule had almost completely disappeared after a week or two. No fundal changes were noted clinically in these eyes; in some examined histologically there were slight changes in the rod and cone layer which may have occurred post-mortem.

In 15 out of the 52 eyes infection was experimentally produced prior to injection of the drug. Histological changes in these cases were possibly products of both the drug and the infection and their significance was considered in the light of the changes found in those eyes into which only the drug had been injected.

COMMERCIAL PENICILLIN

Eight eyes were observed over a period of 10 to 29 days and 4 were sectioned. In only 1 eye had infection been experimentally produced. Opacities, sometimes dense, appeared in the vitreous body and remained until the end of the experiments. These eyes received a single dose of 2000 units or, in one eye, of 5000 units; or a double dose of 2000 units with an interval of 48 hours. The fundi

suffered damage in all cases. Below the medullated nerve fibre bundle pigmentary changes developed, and, in one case, this progressed to a retinitis proliferans with detachment. Three eyes showed fine new vessel formation with swelling of the medullated nerve fibre bundle, and retinal haemorrhages appeared in one of them. Posterior cortical lenticular opacities appeared once.

Histologically, in keeping with these findings, all the sectioned eyes show numerous monocytes in the posterior vitreous, large circumscribed areas of complete retinal destruction with glial replacement, and pigment aggregates in the retina resulting from disturbance of the retinal pigment layer. The vessels, which normally lie free on the medullated nerve fibre bundle, are embedded in a non-cellular matrix. This matrix is possibly a factor in producing the swollen appearance in the nerve fibre bundle seen ophthalmoscopically. No infiltrates or transudates are present within the retina, and the choroid is unchanged.

PURE PENICILLIN

Fourteen eyes were examined over a period of 10 to 27 days and 8 were sectioned. These eyes received a single dose of 2000 units or of 5000 units of crystalline penicillin; or a double dose of 2000 units with an interval of 48 hours. In 4 eyes the penicillin was injected 6 hours after inoculation of the vitreous with haemolytic streptococci. Toxic effects, while present in many of the eyes, were much less than with the commercial preparations. In one eye there was swelling of the medullated nerve fibre bundle and in another opacities in the posterior cortex of the lens. Histologically 3 out of 8 eyes sectioned show areas of retinal destruction. This is in keeping with the findings of Sallmann *et alia* (1944) who found 4 out of 11 eyes so affected. The histological changes are more marked than the clinical appearances had suggested. For example, 2 non-infected eyes in which vitreous opacities were the only ophthalmoscopic changes show circumscribed areas of retinal destruction and in the lower periphery an accumulation of pigment in the rod and cone layer. These eyes received a single dose and a double dose of 2000 units of crystalline penicillin respectively and indicate the difficulty, repeatedly experienced in these experiments, of assessing cellular changes in the retina on the basis of the ophthalmoscopic appearances alone. In one eye there are numerous small pigment aggregates within the retina. The eyes receiving a double dose were more affected than the others. These findings show that crystalline penicillin, in the doses stated, can produce definite toxic effects on the retina. Nevertheless, the changes are not so marked as with the commercial preparation, and unlike the latter, crystalline penicillin appears to have no predilection for the vascular system.

SODIUM SULPHACETAMIDE

Of 16 eyes examined over a period of 10 to 128 days, 13 were sectioned. The eyes received a single dose of 0.1 c.c. of 30 per cent. sodium sulphacetamide or a double dose of that quantity with an interval of 48 hours. In 5 eyes the drug was given at one hour after the inoculation of the vitreous with haemolytic streptococci. Of the 11 eyes in which no infection was introduced the fundi of 6 appeared normal while in the others patchy changes of a mottled appearance developed in the lower part of the fundus. There were no notable changes in the retinal vessels. In all the sectioned globes there are circumscribed areas, varying in size, of complete retinal destruction in the lower part of the fundus, excepting the 5 eyes infected with haemolytic streptococci. As these latter eyes had been removed 10 days after the infection and the minimum period between injection and enucleation in the others was 75 days, it may be presumed that a definite period of probably several weeks is required for the development of areas of complete retinal destruction. The eye which received a double dose of the drug is more affected than the others and shows retinal detachment and cystic formation within the retina as well as an extensive area of destruction. Practically all the eyes (infected and non-infected alike) show fairly widespread changes at the level of the rod and cone layer as indicated by the presence of pale staining detritus and numerous large droplets. These appear to be the product of the rod and cone layer and not of the pigment epithelium as suggested by Koyanagi and Kinnikawa (1937) and von Sallmann *et alia* (1944). This change can be noted particularly in the retina adjacent to the atrophic areas already noted. In assessing the significance of this appearance it must be remembered that it may be noted in a lesser degree as a post-mortem change. In some cases the outer nuclear layer of the portions of the retina affected in this way shows diminution in the number of its cells, and in one case there is complete absence of the outer molecular layer in the area corresponding to the maximum degeneration of the rods and cones. It is noteworthy that the fundi of several of the sectioned eyes showing marked histological changes were clinically normal, as had indeed previously been found by Leopold and Scheie (1943). Beyond 100 days infiltration of the vitreous with monocytes is not a feature of any of the cases.

After 128 days 5 eyes which were stained for Nissl's granules, with Marchi's method and for fat with scarlet red, show no abnormalities in the ganglion cells, in the medullary sheaths or in the nerve fibre bundles. In no case was the choroid found to be affected.

The toxic effects of sodium sulphacetamide on the retina appear to be more severe than those of crystalline penicillin; and further,

there is a difference in that the areas of destruction with sodium sulphacetamide appear only after a delay of several weeks. With both drugs, however, the initial effect is apparently on the outer part of the retina. It is noteworthy that the media, including the lens, were clear in all cases after 128 days.

MARFANIL

Injections of marfanil were made in 5 eyes, 4 of which were sectioned. Each case received a single injection of 0.1 c.c. 30 per cent. solution or two such doses at an interval of 48 hours. In 2 cases the injection was preceded by the inoculation of haemolytic streptococci. The periods of observation were 10 to 17 days.

Vitreous opacities were slight. The most notable clinical features in all cases were constriction and, in places, variation in calibre of the retinal vessels which were often reduced to threads. Two of the cases showed swelling of the medullated nerve fibre bundle. One showed haemorrhages and new vessel formation. In all cases examined histologically there are circumscribed areas of retinal destruction and in many, pigment aggregates within the retina. In 2 cases there appears to be thickening of the retinal vessel walls.

Marfanil is more toxic to the retina than sulphacetamide or penicillin. Like impure penicillin it appears to have an effect on the retinal vessels and to cause pigmentation of the retina.

V.335

Injections of V.335 were made in 9 eyes of which 7 subsequently were sectioned. A single dose, or two doses at an interval of 48 hours, was given and in 2 cases the injection was preceded by experimental infection of the vitreous. The periods of observation were from 10 to 33 days.

Only slight opacities formed in the vitreous. Clinically all eyes had marked changes in the lower part of the fundus with obvious pigment disturbance. The vessels were not notably affected. Histological examination confirms the severity of the fundal changes. In all cases there are extensive areas of retinal destruction and 3 eyes show separation of the retina. In most there are marked disturbances of the rod and cone layer, similar in appearance to those already described. In 3 the choroid shows numerous foci of round cells, though none of these eyes had been infected. Both the clinical and histological findings indicate that V.335 is toxic to the retina and apparently to the choroid. Of all the drugs used and described in this report it appears to have been the most toxic.

Summary of effect of drugs on tissues of rabbits' fundus

(1) A study of these 52 eyes shows that all of the drugs used are capable of producing a toxic effect on the retinal cells. In

Table I the drugs are arranged in the order of the increasing severity of their toxic effects. It is obvious that crystalline penicillin is the least toxic of the 5 substances tested and V.335 the most toxic. Although it is possible to assess the vessel changes clinically, the assessment of retinal cell damage must be based on the eyes histologically examined. The number of eyes showing areas of retinal destruction is therefore expressed as a fraction of the eyes sectioned.

TABLE I

Showing certain toxic effects of drugs arranged in order of their increasing severity.

Drug	No. of eyes observed clinically	No. of eyes showing vessel changes in the fundus expressed as fraction of eyes clinically observed	No. of eyes showing opacity in posterior lens expressed as fraction of eyes clinically observed	No. of eyes sectioned	No. of eyes showing histological areas of retinal destruction expressed as fraction of eyes observed histologically	No. of eyes showing pigment aggregates in retinae expressed as fraction of eyes observed histologically
Crystalline penicillin	14	$\frac{0}{14}$	$\frac{1}{14}$	8	$\frac{3}{8}$	$\frac{2}{8}$
Commercial penicillin	8	$\frac{3}{8}$	$\frac{1}{8}$	4	$\frac{4}{4}$	$\frac{4}{4}$
Sodium sulphacetamide	16	$\frac{0}{16}$	$\frac{0}{16}$	13	$\frac{8^*}{13}$	$\frac{0}{13}$
Marfanil ...	5	$\frac{5}{5}$	$\frac{1}{5}$	4	$\frac{4}{4}$	$\frac{3}{4}$
V. 335 ...	9	$\frac{0}{9}$	$\frac{1}{9}$	7	$\frac{7}{7}$	$\frac{2}{7}$

* The five eyes which do not show areas of retinal destruction were removed 10 days after injection, a period probably too short for development of these areas in the case of sodium sulphacetamide. Such areas of destruction were present in all eyes sectioned after 73 days.

(2) Although with all these drugs eyes are shown in which there are localised patches of retinal destruction in the lower part of the fundus, the initial effect especially with sodium sulphacetamide, appears to be on the rod and cone layer. This is suggested by the

eyes in which the only retinal changes were in the rod and cone layer, the presence of changes in that layer in the otherwise normal retina adjacent to foci of complete retinal destruction, the presence in several eyes of cystic and other changes in the outer nuclear layer, and the absence of changes in the inner retinal elements in several eyes specifically stained. Sallmann *et alia* (1944) have commented on the early involvement of the outer retinal elements following intra-vitreous injection of penicillin.

(3) Commercial penicillin and marfanil are capable of a toxic effect on the retinal vessels as indicated clinically by vascular constriction, calibre variation, and new vessel formation, and histologically in one or two eyes by changes in the vessel walls.

(4) In no case were there transudative or exudative changes present in the retina.

(5) The choroid was free from change in practically all eyes.

(6) Correlation between ophthalmoscopic changes if present and histological findings should be ensured by identification of the lesion in enucleated eyes with the help of the slit-lamp and the binocular microscope. No absolute statement regarding retinal cell changes of even a gross nature can be based on ophthalmoscopic examination alone.

Section II. The diffusion of the drugs within the eye

METHODS

The drugs were injected into the vitreous as described in Section I. After intervals varying from 1 to 72 hours, the animals were killed and certain tissues of the eye were removed for estimation of their drug content.

Sulphacetamide estimations were made on the aqueous, vitreous, cornea, iris, lens, chorio-retinal tissue and sclera, which were removed in that order immediately after the rabbit was killed. Following dissection, the tissues were washed rapidly in saline, dried on blotting paper and, after weighing, thoroughly ground in glass mortars with 2 ml. of 15 per cent. trichloroacetic acid and silver sand. The macerated tissues were allowed to stand for 30 minutes, when the contents of the mortars were washed and filtered into 25 ml. measuring cylinders and the volume made up to 25 ml. Sulphonamide estimations were made by the colorimetric method of Bratton and Marshall (1939) in 10 ml. aliquot portions. Readings were taken on a Klett visual colorimeter and Spekker photo-electric absorptionmeter.

Penicillin estimations were made on the aqueous, vitreous and cornea. These tissues were removed from the rabbit and subsequently handled with aseptic precautions. The cornea was ground in an agate mortar with sand and with a certain measured amount

of nutrient broth; after standing for one hour, the broth containing the macerated cornea was centrifuged and the supernatant fluid taken for testing. The penicillin estimations were made by a serial dilution test; this determined the highest dilution of the eye fluids in nutrient broth which completely inhibited growth of the standard (Oxford) *staph. aureus*. The particular dilutions examined in each instance were chosen according to the penicillin concentration considered likely to be present. The volume of the test mixture was usually 0.4 ml. Each tube was inoculated with a loopful of a 1 in 300 dilution of an eighteen hour broth culture of the standard staphylococcus. After incubation at 37°C. for about eighteen hours, the tubes were examined for growth as shown by the presence of turbidity. The presence or absence of growth was confirmed by a stroke-subculture on a blood agar plate. In control tests, it was found that the lowest concentration of penicillin completely inhibiting growth was between 1/20th and 1/25th of a unit per ml. For the purpose of calculating the penicillin concentrations in unknown fluids in terms of unit per ml., it was assumed that their highest bacterio-static dilutions contained 1/20th of a unit per ml. For instance, if growth of the staphylococcus was inhibited by the fluid diluted 1 in 20, but not by the fluid diluted 1 in 40, the penicillin concentration of the fluid was reported as being between 1 and 2 units per ml. (The eye fluids themselves, in the absence of penicillin, were not found to have any bacteriostatic effect in the dilutions tested).

RESULTS

(a) *Injection of sodium sulphacetamide into the normal vitreous.*—Table II shows the concentrations of sulphacetamide found in the vitreous, aqueous, cornea, iris, lens, chorioretinal tissue and sclera, one hour, six hours, one, two, three and four days after the injection of 0.1 ml. of 30 per cent. sodium sulphacetamide.

It was found that the drug rapidly diffused into all the tissues which always contained high concentrations within an hour after injection. In the vitreous, the concentration fell from about 1000 mg. per cent. (i.e. per 100 gm.) at one hour after injection to an average value of 2.5 mg. per cent. after four days. Chemotherapeutic concentrations (i.e., over 5 or 10 mg. per cent.) were usually found in all the ocular tissues after 2 days and sometimes, in the vitreous, even after 3 days. In Figs. 2 and 3, the logarithm of the average concentration of sulphacetamide has been plotted against the time after injection. In the vitreous (Fig. 2), the log. of the concentration fell in inverse proportion to the time after injection.

(b) *Injection of sodium penicillin into the normal vitreous.*—Table III shows the concentrations of penicillin found in the vitreous, aqueous and cornea, one hour, six hours, one day, two days and

three days, after the injection of 2000 units of impure sodium penicillin into the vitreous. Table IV shows the results for similar experiments with pure crystalline sodium penicillin.

TABLE II

Concentrations of sulphacetamide in the ocular fluids and tissues after the intra-vitreous injection of 0.1 ml. 30% sodium sulphacetamide in the normal eyes of rabbits.

Time	Rabbit No.	Mg. Sulphacetamide per 100 g.						
		Vitreous	Aqueous	Cornea	Iris	Lens	Choroid and Retina	Sclera
1 hr.	1896 R.E.	1300	232	102	122	21.1	290	213
	1898A "	1300	69.3	51.6	183	28.3	250	212
	1910A { R.E.	960	268	177	308	—	391	149
	{ L.E.	980	342	257	440	—	351	198
6 hrs.	1896 L.E.	670	—	105	138	38.0	120	115
	1898A "	1080	218	180	263	61.7	254	121
	1911A { R.E.	550	205	187	221	—	202	125
	{ L.E.	810	275	203	253	—	287	197
1 day	1888 { L.E.	209	92.5	94.0	106	—	—	71.7
	{ L.E.	353	111	84.2	12.0	—	—	43.7
	1900A { R.E.	310	49.9	70.0	36.6	38.5	41.0	—
	{ L.E.	311	58.2	64.7	85.0	20.2	42.5	45.0
	1912A { R.E.	201	20.5	20.7	—	—	—	—
	{ L.E.	239	63.5	80.1	74.5	—	—	—
2 days	1887 { R.E.	115.1	18.9	11.3	19.2	—	—	38.2
	{ L.E.	70.9	—	7.0	14.0	—	—	10.1
	1901A { R.E.	31.1	4.0	6.3	11.1	16.9	10.7	13.5
	{ L.E.	25.7	3.5	7.4	12.1	13.9	9.6	7.9
3 days	1886 { R.E.	23.7	4.9	2.8	3.0	—	—	2.0
	{ L.E.	13.0	0.85	1.0	1.6	—	—	1.2
	1902A { R.E.	1.5	0.5	1.0	1.0	15.2	—	1.0
	{ L.E.	0.9	1.0	0.5	1.0	9.0	—	1.0
4 days	1903A { R.E.	3.4	0.6	0.6	—	—	—	—
	{ L.E.	6.3	0.9	0.6	—	—	—	—
	1909A { R.E.	0.1	0.2	0.5	—	—	—	—
	{ L.E.	0.5	0.2	0.5	—	—	—	—

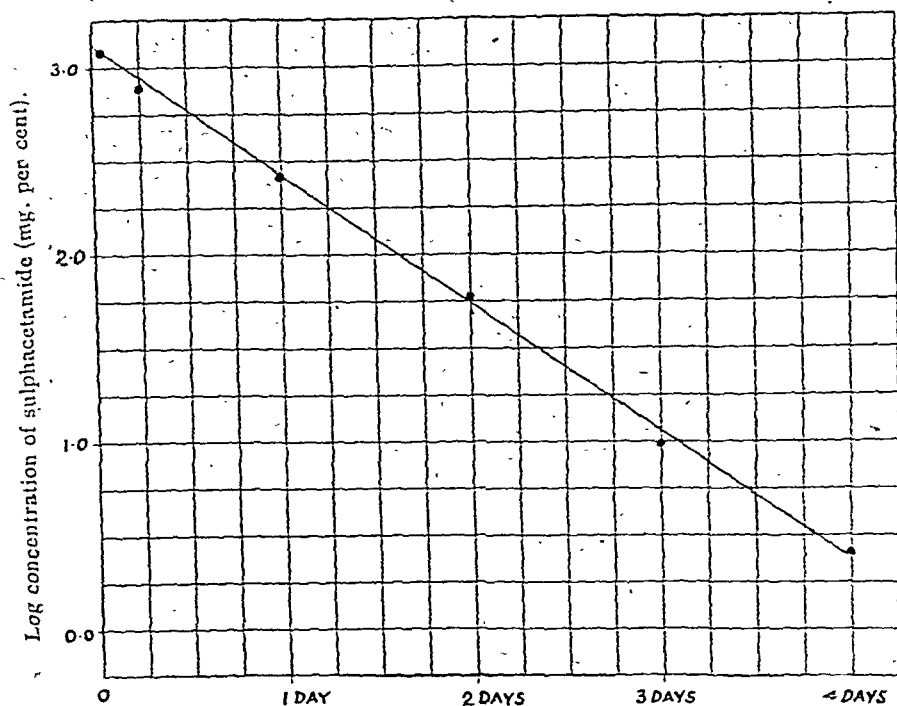


FIG. 2.

Persistence of sulphacetamide in the vitreous following intra-vitreous injection of 30 mg. sodium sulphacetamide.

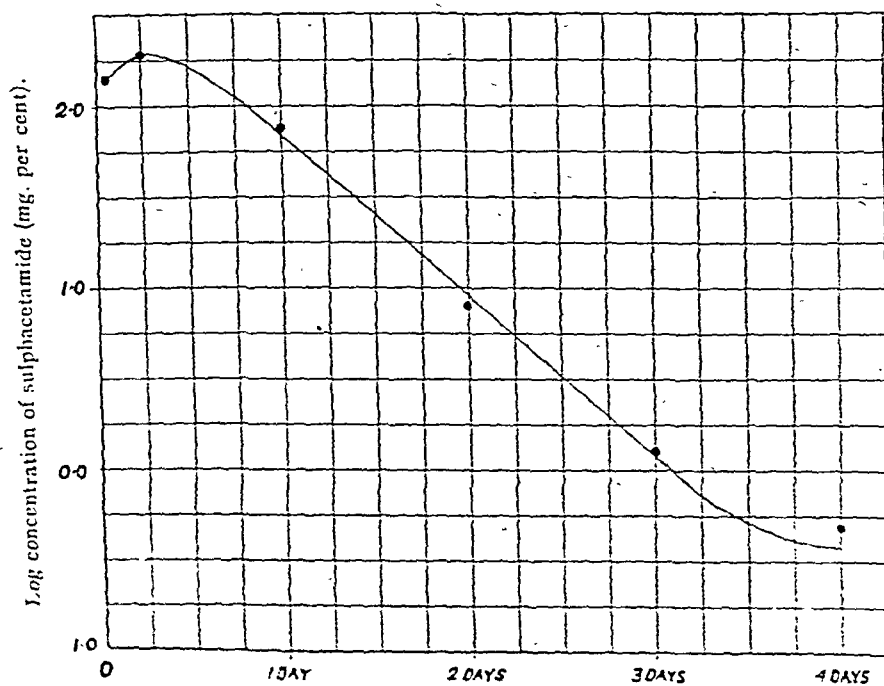


FIG. 3.

Persistence of sulphacetamide in the cornea following intra-vitreous injection of 30 mg. sodium sulphacetamide.

TABLE III

Concentration of penicillin in the aqueous, vitreous and cornea, 1 hour, 6 hours, 1, 2, and 3 days after the intra-vitreous injection of 2000 units of impure sodium penicillin.

Time	Rabbit No.	Concentration of Penicillin (units per ml.)		
		Vitreous	Aqueous	Cornea
1 hour	1892 R.E.	500-1000	20-40	16-32
6 hours	1892 L.E.	500-1000	5-10	8-16
1 day ...	1873	R.E. 100-300	—	—
		L.E. 100-300	—	—
	1891	R.E. 100 200	3-6	2-4
		L.E. 200-400	6 12	4-8
	1893	R.E. 200-400	1-2	2-4
		L.E. 200-400	1-2	2-4
2 days	1874	R.E. 6-10	—	—
		L.E. 6-10	—	—
	1890	R.E. <1/5	<1/10	<1/2
		L.E. 1-2	1/10-1/5	<1/2
	1894	R.E. 5-10	1/10-1/5	} <1/4
		L.E. 1-2	1/10	
3 days	1889	R.E. <1/10	<1/10	<1/2
		L.E. <1/10	<1/10	<1/2
	1895	R.E. 1/10-1/5	<1/10	} <1/4*
		L.E. 1/10-1/5	<1/10	

*Tissues from both eyes analysed together.

It was found that penicillin diffused rapidly from the vitreous into the aqueous and cornea which always contained high concentrations within an hour after injection. The concentration of penicillin in the vitreous remained, however, much greater than the concentrations in the aqueous and cornea; the vitreous thus acts as a depot replenishing the drug lost from the other tissues. The concentration of penicillin fell from the high levels at one hour after injection to levels approaching or below the limits of detection after two days in the case of the vitreous. Chemotherapeutic concentrations (*i.e.*, about 1/20th of a unit per ml. and over) were maintained in the vitreous for 2 to 3 days, and in the aqueous and cornea for 1 to 2 days.

TABLE IV

Concentrations of penicillin in the aqueous, vitreous and cornea, 1 hour, 6 hours, 1, 2 and 3 days after intra-vitreous injection of 2000 units of pure sodium penicillin.

Time	Rabbit No.	(Concentration of Penicillin (units per ml.))		
		Vitreous	Aqueous	Cornea
1 hour	1922	R.E. 1000-2000	4-8	4-8
		L.E. 2000-4000	2-4	8-16
	1934	R.E. 500-1000	2-4	1-2
		L.E. 500-1000	2-4	2-4
	1938	R.E. —	25-50	—
		L.E. —	—	—
6 hours	1921	R.E. 500-1000	8-16	4-8
		L.E. 1000-2000	2-4	4-8
	1935	R.E. 1000-2000	8-16	8-16
		L.E. 500-1000	8-16	8-16
	1939	R.E. —	1/2-3	—
		L.E. —	3-6	—
1 day	1920	R.E. 6+128	<1/4	<1/2
		L.E. 32-64	1/4-1/2	1/2-1
	1936	R.E. 16-32	1/4-1/2	1/2
		L.E. 32-64	1/2-1	1/2-1
	1940	R.E. —	1/2-1	—
		L.E. —	—	—
2 days	1919	R.E. 1/2-1	<1/10	<1/4*
		L.E. 1/2-1	<1/10	<1/4*
	1937	R.E. 1-2	1/10-1/5	<1/4*
		L.E. 1-2	1/10-1/5	<1/4*
3 days	1918	R.E. <1/10	<1/10	<1/4*
		L.E. <1/10	<1/10	<1/4*
	1938A	R.E. <1/10	<1/10	<1/4*
		L.E. <1/10	<1/10	<1/4*

* Tissues from both eyes analysed together.

In Figs. 4 and 5 the logarithm of the average concentration of penicillin has been plotted against the time after injection. It will be noted that, in the case of pure penicillin, except for a short period immediately after the injection, the logarithm of the

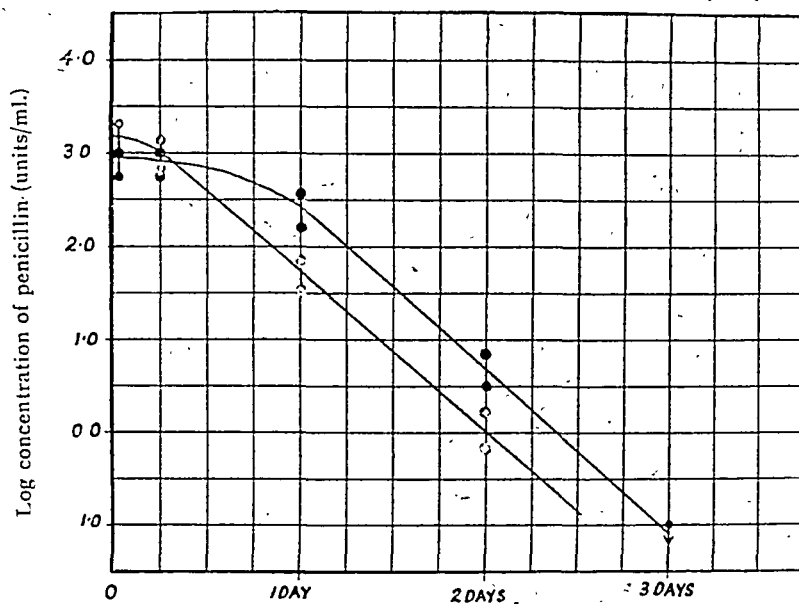


FIG. 4.

Persistence of penicillin in the vitreous following the intra-vitreous injection of 2000 units of penicillin.

● Impure penicillin.

○ Pure sodium penicillin.

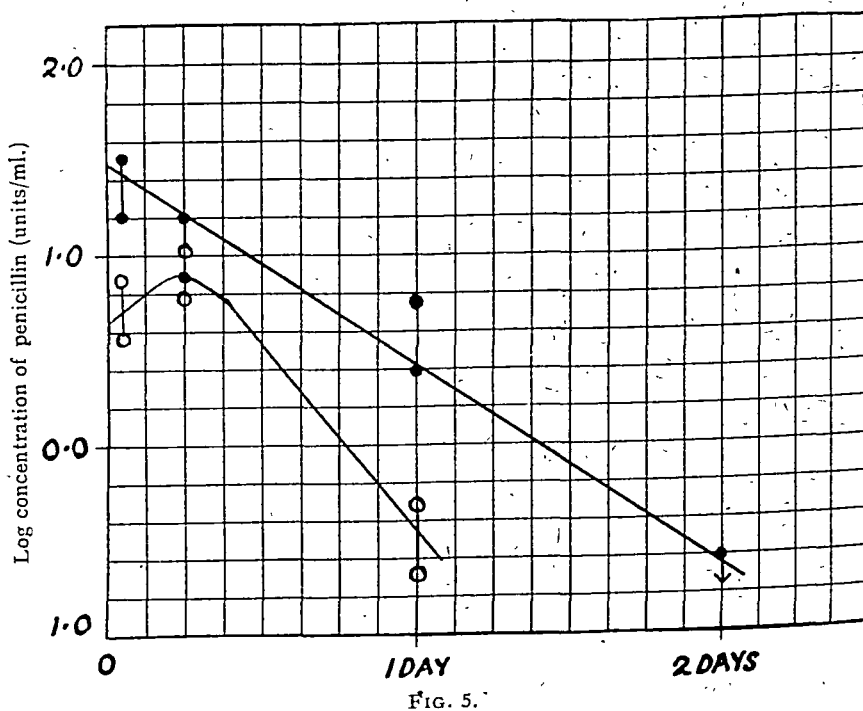


FIG. 5.

Persistence of penicillin in the cornea following the intra-vitreous injection of 2000 units of penicillin.

● Impure penicillin.

○ Pure sodium penicillin.

concentration diminishes in inverse proportion with the time after injection. In the case of impure penicillin, the fall-in concentration is somewhat delayed during the first twenty-four hours.

(c) *Injection of sodium sulphacetamide and sodium penicillin combined into the normal vitreous.* Experiments *in vitro* showed that incubation of sodium penicillin with sodium sulphacetamide for 24 hours at 37°C. did not destroy any appreciable amount of penicillin.

Table V shows the results of experiments in which 0.1 ml. of 30 per cent. sodium sulphacetamide containing 2000 units of pure

TABLE V

Concentrations of penicillin and sulphacetamide in the ocular fluids and tissues after the intra-vitreous injection of 2000 units pure sodium penicillin, and 0.1 ml. 30 per cent. sodium sulphacetamide combined, into normal eyes.

Time	Rabbit No.	Concentration of penicillin (units/ml.)			Concentration of sulphacetamide (mg./100s.)				
		Vitreous	Aqueous	Cornea	Vitreous	Aqueous	Cornea	Iris	Sclera
1 day	1941	R.E. 16-32	$\frac{1}{2}$ -1*	1	145	{ 43.5	47.8	61.1	17.9
		L.E. 16-32	$\frac{1}{2}$ -1*	1-2	165		55.3	75.2	107
2 days	1942	R.E. $<\frac{1}{2}$	$<1/10^*$	$<\frac{1}{2}^*$	3.9	{ 2.5*	—	—	8.0
		L.E. $\frac{1}{2}$ -1	$<1/10^*$	$<\frac{1}{2}^*$	37.7		0.2	0.9	1.8
	1946	R.E. 6-12*	$<1/10^*$	$<\frac{1}{2}^*$	55.1	{ 2.9*	2.1*	6.4	—
		L.E. 6-12*	$<1/10^*$	$<\frac{1}{2}^*$	61.4		6.1	—	—

* Tissue of both eyes analysed together.

sodium penicillin was injected into the vitreous. Except in the case of one rabbit (1942) which was examined at 2 days after injection, when low drug concentrations were found, the results were similar to those obtained when the drugs were injected singly. The combined administration of sulphacetamide and penicillin by intra-vitreous injection does not seem, therefore, to be contra-indicated by any increased rate of disappearance of either drug.

(d) *Injection of sodium sulphacetamide and sodium penicillin into the infected vitreous.* Table VI shows the results of experiments in which the drugs were injected both together and singly into the vitreous of the eyes of rabbits infected by intra-vitreous injection of haemolytic streptococci. Rabbit 1945, at 2 days after injection with penicillin and sulphacetamide combined, showed drug concentrations in the ocular tissues similar to those found in experiments with non-infected eyes. Penicillin alone was injected into the eyes,

TABLE VI

Concentration of penicillin and sulphacetamide in the ocular fluids and tissues after the intra-vitreous injection of 2000 units pure sodium penicillin and 0.1 ml. 30 per cent. sodium sulphacetamide combined, and singly, into infected eyes.

Time	Rabbit No.	Condition	Concentration of penicillin (units per ml.)			Concentration of sulphacetamide (mg/100 g.)			
			Vitreous	Aqueous	Cornea	Vitreous	Aqueous	Cornea	Iris
1 day	1943	R.E. Infected	1-1	<1/5	<1	9.3	5.7	2.5	1.9
		L.E. Infected			perforated				
	1951	R.E. Infected	50-100	1/5-1	1-2	—	—	—	—
		L.E. Normal	50-100		contaminated	—	—	—	—
2 days	1960	R.E. Infected	—	—	—	56.6	5.4	2.0	8.1
		L.E. Normal	—	—	—	322	52.3	46.3	62.2
	1945	R.E. Infected	2.4*	1/10*	<1*	112	20.7*	19.2*	32.2
		L.E. Infected				8.2			21.0

* Tissues from both eyes analysed together.

one infected and one normal, of rabbit 1951; after one day the penicillin concentrations in the vitreous fluids of the two eyes were similar. On the other hand, in rabbits 1943 (injected with penicillin and sulphacetamide combined), and 1960 (injected with sulphacetamide alone) the drug concentrations at one day after injection were considerably lower in the infected eyes than in normal eyes. It appears that in some cases of infection, disappearance of the drug from the eye is accelerated, but even so, chemotherapeutic concentrations are maintained for about one day after intra-vitreous injection.

Section III. Effect of Drugs on experimentally produced infections of the Vitreous Body

METHODS

In all animals both eyes were infected, one eye in each animal was treated, while the other served as control and received an injection of saline. Intra-ocular infection was produced, by the injection into the vitreous, of virulent haemolytic streptococci, the strain (1c) being that previously used in the production of corneal lesions (Robson & Scott, 1944); 0.02 c.c. of a broth culture diluted 1:1000 was the standard dose and was followed by the therapeutic agent, after an interval of 1, 6 or 24 hours. In a few experiments a second injection of the drug was given 48 hours later. The dose was always

0.1 c.c. of a 30 per cent. solution, except for penicillin, when the concentration was 20,000 units c.c. At the end of ten days the animals were killed, cultures were taken from the vitreous and some of the eyes were sectioned.

RESULTS

In untreated eyes signs of a severe reaction were evident within 24 hours of inoculation. There was a severe uveitis, shown by exudates into the vitreous and acute iritis. Twenty-four hours later, the whole vitreous was usually opaque and hypopyon was frequently present. At the end of the experiment there was a vitreous abscess in most of these eyes, and in the remainder the vitreous was completely opaque. The effects of treatment are shown in Table VII.

TABLE VII

Showing the effect of various drugs on the development of vitreous infections.

Drug	Number of Animals	Interval between inoculation and treatment	Result treated eyes
		<i>Hours</i>	
Penicillin	3	1	No infection
Penicillin	1	6	No infection
Pure penicillin	2	6	No infection
Pure penicillin	3	24 and 72	As controls
Sodium sulphacetamide	4	1	No infection
Sodium sulphacetamide	2	1	Infection delayed
Pure penicillin and sodium sulphacetamide	3	24 and 72	As controls
Marfanil	2	1	No infection
Marfanil	2	6	No infection
V.335	2	1	No infection

It will be seen that penicillin prevented the development of infection when injected up to 6 hours after inoculation but had no effect when the treatment was delayed for 24 hours, even if the drug was combined with sodium sulphacetamide.

Sodium sulphacetamide alone injected 1 hour after inoculation prevented development of infection in some of the cases—merely delaying it in others.

Marfanil and V. 335 gave favourable results, but in view of their obvious toxicity to ocular tissues, no further attention was given to them.

The cultures made from the vitreous of the untreated eyes showed growth of haemolytic streptococci in about 50 per cent. of cases but

in the remainder the abscess had apparently become sterile and the cultures were negative. No haemolytic streptococci were recovered from any of the treated eyes.

In a number of the experiments in which the infection had obviously been controlled, certain effects were noted in the vitreous and retina of treated eyes which, in the light of the experiments described in Section I, could, with confidence, be attributed to the toxic effects of the drugs.

Discussion

An attempt has been made to investigate the value and the practicability of injecting drugs intra-vitreously.

Ophthalmologists, understandably, are hesitant to use this method on account of the possible damage to the vitreous gel, the retina, etc. Experimentally, using no surface diathermy, a small needle could be introduced into the vitreous and a small quantity of saline injected without any ill effects. With the exception of pure penicillin, all the drugs introduced into the vitreous, including impure penicillin, were highly damaging to the retina, to a degree which excludes their use clinically. Pure penicillin in some cases was shown to cause small areas of retinal damage—but slight by comparison with impure penicillin or any of the other drugs. Under favourable conditions penicillin introduced by this means is highly effective in the control of infections of the vitreous body, and the diffusion experiments demonstrate that the drug diffuses but slowly from it, so maintaining a chemo-therapeutic concentration for 2-3 days after a single injection, in contrast to its rapid disappearance when injected into most other tissues in the body. In cases in which an eye is seriously endangered by infection in the vitreous it appears justifiable to consider the use of pure penicillin by intra-vitreous injection—the control of the infection more than outweighing the possibility of small areas of retinal damage. If doses comparable to those used in the experiments are given, it would appear sufficient to repeat the injections at intervals of two to three days; the slow diffusion from the vitreous permitting a therapeutic level of the drug to remain for that time.

Summary

The toxic effects and rate of diffusion of certain drugs introduced into the vitreous of rabbits have been investigated.

The therapeutic value of these drugs in experimentally produced vitreous infections is recorded.

Impure penicillin, sodium sulphacetamide, marfanil and V.335 do not appear to have a clinical value by intra-vitreous injection because of the damage they do to the retina.

Pure sodium penicillin sometimes damages the retina slightly when injected intra-vitreously, but its use by this means seems indicated and justifiable in certain cases of infection.

We are very grateful to Dr. W. B. Levinthal for his help in this work and to the W. H. Ross Foundation (Scotland) for the Prevention of Blindness, who defrayed the expenses. The pure penicillin was kindly supplied by Glaxo Laboratories Ltd., the sodium sulphacetamide (Albucid soluble) and marfanil by British Schering Ltd., and the V.335 by the Boots Pure Drug Company Ltd.

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A CASE OF THE LAURENCE-MOON-BIEDL SYNDROME SHOWING ATYPICAL RETINITIS PIGMENTOSA ASSOCIATED WITH MACULAR DYSTROPHY*

BY

CHARLES TAYLOR

LONDON

IN their comprehensive studies on the Laurence-Moon-Biedl syndrome, Cockayne, Krestin and Sorsby (1935), and Sorsby, Avery and Cockayne (1939) have shown that the fundus lesion may be an atypical rather than typical retinitis pigmentosa, and that macular dystrophy with optic atrophy may replace either of these, an observation also made more recently by Lyle (1946). The present case report is of interest in that it shows the combination of atypical retinitis pigmentosa with macular dystrophy. The great rarity of the association of retinitis pigmentosa and macular dystrophy and its hereditary character have been brought out by Sorsby (1940 and 1941).

Case report

Past History. T. S., aged 24 years, was fat at birth. There was some loss of weight following illness at 3 weeks (said to be "threatened with meningitis"). No attempt was made at talking

* Received for publication, January 29, 1947.

until his fifth year when he was sent to school. During his first year at school lenses were prescribed by an ophthalmologist and during his school life he has always occupied the front bench. As far as he can remember there has always been difficulty in getting around after dusk. He rode a bicycle until he was twelve years-old, but gave up cycling when he collided with a woman who was crossing the road. In the same year his master forbade him to play cricket. His education was carried on at a private (not "special") school until he was sixteen years of age when it seemed hopeless to continue. He had a variety of posts which he could not retain and

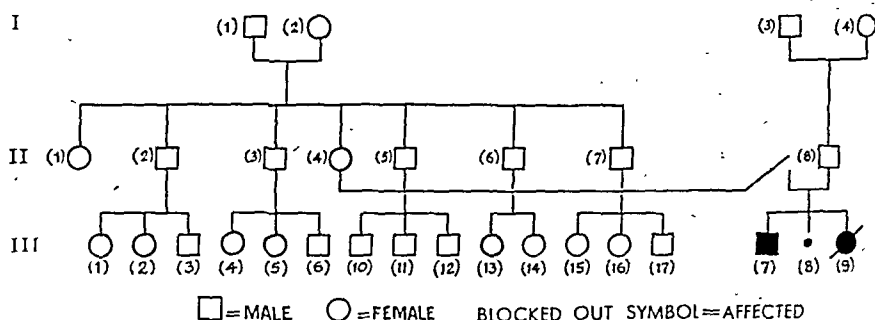


FIG. I. (Three generations only).

- II (1) Died in infancy from diphtheria.
 (2) Died of sarcoma of spine at 38 years.
 (3) (6) (7) Short-sighted.
- III (5) Tuberculosis of hip.
 (7) Patient (T. S.).
 (8) Miscarriage (•).
 (9) Still-born, affected.
- (14) Died at 3 years from diphtheria; had cleft palate.

is now employed in a factory, where, by pulling a lever pieces are punched out in a sheet of metal to retain hypodermic needles.

Family history. (Fig. 1). His father and mother are not related. They are both English. The father has always enjoyed good health but the mother has had two nervous breakdowns. The Wassermann reaction in both of them was negative. On the father's side of the family a brother and sister of the great grandmother died in infancy, and a great aunt had several miscarriages and two children who died at one year old, one of which was said to have been born blind.

On the mother's side of the family two children of a great aunt died as babies and one great aunt was very fat.

T. S. was the first born. Twice during pregnancy abortion was threatened. A year later there was a four months' miscarriage, but no details of the foetus are available. The third pregnancy resulted in a stillborn female child with extra fingers and toes and "part of the head missing."



FIG. 2.

Examination. T. S. is placid and co-operative. Mental retardation is present in only a minor degree and in spite of his visual disability he has a fair knowledge of recent events. He is distinctly obese (Fig. 2). His height is 5 ft. 6 in. and his weight 14 st. 2 lb. The fat is distributed chiefly on face, chest, abdomen and thighs. His hair is dark. He presents the male distribution of hair on the body and has to shave every day. The testicles are descended and within normal limits of size. The penis is undersized and the voice high-pitched.

Scars are present on the ulnar side of the right hand over the head of the fifth metacarpal and on the outer side of each foot over the heads of the fifth metatarsals. The extra digits were removed in childhood. Dorsal scoliosis and genu valgum are present.

Vision of each eye with correction:—

$$\left(\begin{array}{l} \text{R. } -1.50 \text{ D. sph.} \\ \quad -2.0 \text{ D. cyl. } 60^\circ \end{array} \quad \text{L. } -1.0 \text{ D. sph.} \\ \quad -2.0 \text{ D. cyl. } 180^\circ \right)$$

and without correction is 2/60 after much searching. There is complete and absolute loss of the peripheral fields and as far as can be ascertained only small receptive areas in the perimacular regions remain. The left eye is divergent 5-10 degrees. Both pupils are equal and react to light and convergence accommodation. Constant

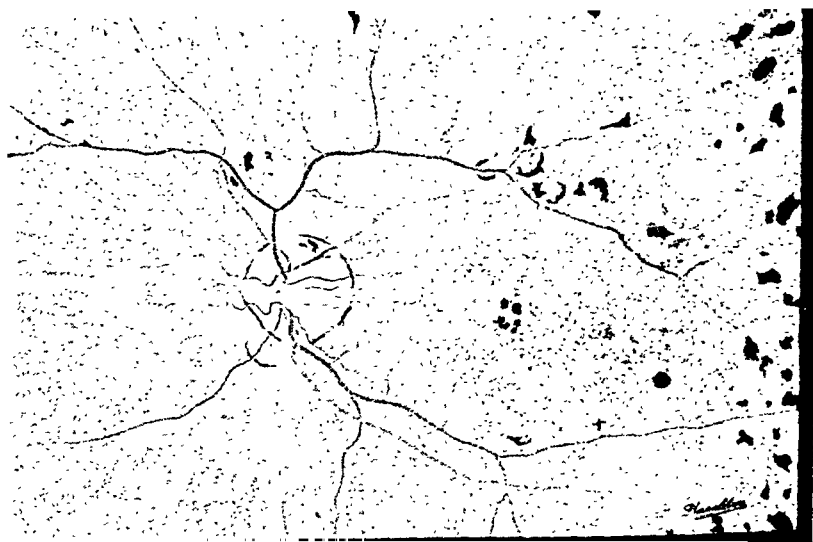


FIG. 3.

Left fundus of T. S.

bilateral nystagmus of the pendulum type is present. It is slow in rate, and increased in range on lateral fixation. A small posterior polar cataract is present in each eye. The fundi (Fig. 3) are lack-lustre. Small patches of pigment of varying shapes are scattered over the fundi but "bone corpuscle" pigmentation is not predominant. In addition there is a fine peppering of pigment where the tapeto-retinal atrophy is more marked. Occasional small circumscribed areas of retino-choroidal atrophy are present. The maculae are spotted with pigment. The retinal vessels are diminished in calibre. Unmasking of the choroidal vessels is present over the greater part of both fundi except in the macular and perimacular regions.

Motor and sensory functions of the cranial nerves (apart from 2nd nerves) are normal.

Muscle tone and power in the limbs is good. There is no ataxia. Deep and superficial reflexes are normal and sensation good.

Examination of heart, lungs, and abdomen reveals no abnormality.

Blood pressure: Systolic 128. Diastolic 87. *Urine:* No abnormality. No polyuria or polydipsia. *Blood:* Wassermann reaction: negative. X-ray of skull shows normal conformation. The sella turcica shows no abnormality. X-ray of hands and feet: nil abnormal.

Remarks

Reviews of the collected families present a large number of miscarriages and early deaths indicating that the expression of the genes may be so severe as to result in non-viability or early death. In this sibship it may be that the miscarriage was due to this cause and there is little doubt that this accounts for the still birth of the affected female child.

The present case taken in connection with the previously reported cases, indicates that the range of fundus lesions in the Laurence-Moon-Biedl syndrome extends to at least four variants of tapeto-retinal degeneration: typical retinitis pigmentosa, atypical retinitis pigmentosa, macular dystrophy, and atypical retinitis pigmentosa combined with macular dystrophy.

I am indebted to Mr. L. H. Savin for the opportunity to report the case, and to Professor Arnold Sorsby for his kind assistance.

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THE ACTION OF SULPHANILAMIDE ON RABBITS' LENSES *IN VITRO**†

BY

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GRONINGEN

Introduction

IN a communication published some years ago¹ I described a number of introductory experiments which had the object of showing that the perfusion culture method of de Haan¹⁴ was excellently serviceable for keeping alive rabbits' lenses *in vitro* for many days. This was the first time that it seemed possible to preserve that extraordinarily vulnerable tissue in such a good condition outside the body. All attempts made in that direction had always miscarried until then¹¹.

These preliminary experiments convincingly demonstrated the usefulness of this method. Of course good results can only be obtained when the lens is not injured during the operation.

One of the most striking advantages of the perfusion culture method is the *continual* renewal of the nutrient medium. This liquid, obtained from the abdominal cavity of rabbits and in its composition resembling aqueous humour very much, passes the explants in an uninterrupted current. Probably it is for that very reason that I succeeded in obtaining such good results.

The more important facts of my research work, showing evidence of the high vitality of the explanted lenses, may be summarized as follows:—

1. Unaltered metabolism even in explants several days old. The lenses maintain their normal transparency for many weeks.

2. Lenses of young animals show symptoms of growth, apparent from mitotic cell divisions in the equator region.

3. Ascorbic acid can freely diffuse through the capsule in both directions. When the nutrient liquid contains an adequate quantity of this vitamin its concentration in the lens has normal values too². This indicates that the permeability of the capsule of the explanted lenses does not change.

4. Regeneration of experimentally wounded capsules starts and goes on *in vitro* in exactly the same way as *in vivo*. The final result is in both circumstances a newly formed capsule³.

In subsequent experiments not only the lens but also other ocular tissues, such as the iris, the choroid, the retina and the ciliary body were explanted with equally good results⁴. This enlargement of the

* From the Histological Institute, University Groningen (Director: Professor J. de Haan, M.D.).

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usefulness of our culture method enabled us to investigate the influence of radiant energy on the crystalline lens, covered or not by the iris⁵. The conception of Vogt and co-workers¹⁵ that the lens absorbs the light rays of long wave-lengths in large quantities could be refuted. My experiments showed that the explanted lens only became cataractous under the influence of heat rays, when it was covered by the pigmented iris. Thus my experiments agreed fully with those of Goldmann¹³. Although Goldmann and I worked along quite different lines, we both came to the conclusion that the lens is *indirectly* burnt by the heat rays absorbed in the pigmented iris.

This short enumeration of the results obtained with the explanted rabbits' lenses may give an impression of the possibilities of this culture method.

Subsequently I carried out further investigations with the object of determining whether this method could be made subservient to testing drugs on their toxicity. I chose sulphanilamide (prontosil album), as this drug is easily soluble in water and as it was used in our laboratory to fight against probable infections of the nutrient liquid.

There was still another reason to investigate the influence of sulphanilamide on the lens, as it is known that this drug inhibits the enzyme action of carbonic anhydrase. This enzyme (discovered by Brinkman and Margaria¹²) catalyzes the reaction $\text{H}_2\text{CO}_3 \rightleftharpoons \text{CO}_2 + \text{H}_2\text{O}$. Now the lens contains a very high concentration of this enzyme and as I could show⁶ that in cataractous lenses the concentration of carbonic anhydrase decreases considerably, it is probable that an intimate relation exists between this enzyme and the transparency of the lens. In other words the behaviour of the explanted lens under the influence of sulphanilamide can enable us to get an impression of the toxic action of this drug.

A simple method to determine the concentration of carbonic anhydrase in lenses (and in other ocular tissues) is described in a preceding article⁷. Two later papers deal with some activators and inhibitors of this enzyme^{8, 9}.

For the details of this culture method and the technique I must refer to my former publications.

At the end of each experiment the enzyme concentration and the content of sulphanilamide in the lenses is determined. Sometimes the lens is microscopically examined, this, however, does not yield any important data.

Experimental results

Introductory experiments taught us that a concentration of 5 mgr. per cent. of sulphanilamide in the nutrient fluid (a concentration lower than the blood concentration in the usual therapeutic dose)

was quite harmless for lenses of adult rabbits. In eight day old explants the lenses did not show any opacities (perfusion velocity of the nutrient fluid 1 drop in about 50 to 60 seconds).

A content of 20 mgr. per cent. of sulphanilamide was not noxious to lenses of a seven week old rabbit. After a stay of three days in the culture vessel the lenses were still quite clear.

When, however, the concentration was raised to 400 mgr. per cent. the lenses of a four week old rabbit got very opaque in the short time of two hours. After 15 hours transparency was so much impaired that letters on an underlying paper could no longer be read. After three days the culture was stopped. The opacities were especially localized in the posterior cortex, but also the anterior cortex showed some of them. The lenses contained 385 mgr. per cent. of sulphanilamide, demonstrating that the capsule is freely permeable to this drug. The enzyme action was reduced to $1/5$ of its original value.

Though the deleterious influence to lenses of very young animals was evident, the toxicity of sulphanilamide for lenses of adult rabbits was less pronounced. It was not until after five days that lenses of the latter in the same concentration (400 mgr. per cent.) showed some opacities. After eight days the enzyme action in these lenses was reduced to less than $1/6$ of its original value.

Conclusions

The perfusion culture method proved to be able to give reliable information on the toxic action of chemicals. With the aid of the same method too, I was formerly able to give an affirmative answer to the question whether galactose has a *direct* noxious influence on the lens¹⁰.

The present experiments have shown that sulphanilamide has not any injurious effect on rabbits' lenses when the concentration is not raised above the level reached in normal therapeutic doses. Yet, some caution is advisable in using this drug. Mrs. Schappert-Kimmijser told me (verbal communication) that in a 43 year old patient, suffering from erysipelas faciei and treated with prontosil for no less than 4 months, cataractous changes in both lenses of capsular and sub-capsular character developed.

There was a striking difference between the behaviour of young and old lenses in my experiments. The younger the animals were the more their lenses were susceptible to the noxious influence of sulphanilamide.

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A CASE OF CONGENITAL VERTICAL SHORTNESS OF THE LIDS COMBINED WITH TETRASTICHIASIS*

BY

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CONGENITAL shortness of the lids was extensively studied by E. Fuchs^{1,2} in 1885. Congenital absence (ablepharon) or maximal shortness (microblepharon) of the lids has been observed repeatedly, partly in monsters, and also in otherwise normal individuals (cf. Manz, *Handbuch. d. Ophthalm.*, *Graefe-Saemisch.*, Vol. II, p. 103). Stellwag described mainly the smallness of the lids in the horizontal diameter and the ensuing shortness of the palpebral fissure in this direction but not what, in the opinion of the writer, should be called vertical shortness of the lids. It was left to Fuchs to coin the expressions "height of the lid" and "vertical extension of the skin of the lid". "Height of the lid" is the maximum distance between the lid margin and the centre (*i.e.* half the breadth) of the eyebrow. In measuring this height the individual is asked to close the eyes

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lightly as though he were sleeping. "Vertical extension of the lids" is the maximal distance measured between the free lid margin and the centre of the eyebrow while the lid is held by the cilia and maximally stretched. These two measurements can be obtained only for the upper lid as there is no fixed demarcation between the lower lid and the skin of the cheek and no reference point exists for measuring the lower lid. The proportion between height and vertical extension of the lid is the co-efficient. Once this co-efficient falls below a certain value (1.5) the lid closure becomes abnormal: Normal closure of the lids is possible only if the height of the stretched upper lid is greater by at least one half than the height in a lightly closed state. The co-efficient varies from 1.5 in infants to 1.9 in old people. When the lid closure is not complete, lagophthalmos results. Complications of this condition are conjunctivitis and blepharitis (usually ulcerative). The cornea is rarely involved.

Von Herrenschwand³ published a case of congenital (vertical) shortness of the lids with ectropion of the palpebral conjunctiva. The lid margins were covered with conjunctiva, there were only a few eyelashes present and the lacrymal puncta were absent. The tarsi were normal.

Urmutzer⁴ presented a case of abnormal (vertical) shortness of the lids with marked hyperaemia of the lid margins, madarosis and a slight ectropion of the lower lid. Complete closure of the lids was impossible and the stretched lids were hardly longer than the relaxed ones.

The following case was observed at our out-patient department.

L. J., a boy aged 16 years, showed with closed lids a lagophthalmos of about 2.5 millimetres in both eyes (Fig. 1). The height of the



FIG. 1.

lids was 24 millimetres, the vertical extension 30 millimetres. The co-efficient was, therefore, 1.24. In both upper lids the cutaneous part of the lid margin was slightly everted, particularly in the temporal portion (Fig. 2). There were in both upper lids four rows



FIG. 2.



FIG. 3.

of equally well developed eyelashes. In the palpebral conjunctiva of both upper lids a fine dark line was visible in the subtarsal sulcus from which thin vertical streaks were extending (Fig. 3).

Visual Acuity: R.E. + 0.5 D. sph. c. + 0.5 D. cyl. 180° 5/5 (partly); L.E. + 0.5 D. sph. c. + 0.5 D. cyl. 180° 5/5 (partly).

There was no irritation of the lid margins or the conjunctiva. Apart from an acute blepharo-conjunctivitis in 1939, and a sty in 1941 no inflammatory conditions of the lids had occurred until now.

Discussion

To explain congenital distichiasis Kuhn⁵ assumes a heterologous developmental anomaly. Cilia develop instead of Meibomian glands not only at the anterior but also at the posterior edge of the lid margin, owing to an abnormal preformation of unknown origin. Brailey⁶ is also of the opinion that the epithelial invagination of the material destined to form the Meibomian glands failed to take place and that eyelashes developed in their stead, corresponding exactly to the ducts of the glands. Erdmann⁷ conversely, considers the glands which belong to the posterior row of cilia as a formation of

cilia and Meibomian glands from the same *anlage*, differentiation having failed to take place owing to an unknown cause. According to him the epithelial cone constituting the first *anlage* of both organs may develop into either an eyelash or an acinus of a Meibomian gland. An increase in the number of cilia, polytrichiasis or hypertrichosis, may be congenital or acquired. In congenital polytrichiasis the cilia are arranged in two or even three or four rows instead of only one, conditions which are called distichiasis, tristichiasis or tetrastichiasis⁸. Fuchs⁹ reserves the term distichiasis for those rare cases where there are congenitally two regular rows of cilia in an otherwise normally developed, not inflamed lid. Aubaret¹⁰ also considers congenital distichiasis to be a rare anomaly.

The case presented above shows in addition to congenital vertical shortness of the lids, the rare picture of tetrastichiasis. The dark lines visible in the sulcus subtarsalis seem to be the hair follicles of the supernumerary cilia which have developed instead of the Meibomian glands. In spite of the existing shortness of the lids and the resulting lagophthalmos, the lid margins are altogether free from irritation, possibly because the cilia are regular and well developed. According to Schreiber⁵ the case of congenital symmetrical shortness of the lids with congenital symmetrical ectropion of the lid conjunctiva described by von Herrenschwand³ is unique in the literature. In the present case the partial eversion of the upper lid represents an abortive form of this condition.

While almost all cases of the Fuchs^{1,2} showed inflammatory changes of the conjunctiva and the lid margins, in the case described here there were no signs of irritation. The cases of von Herrenschwand³ and Urmutzer⁴ had scanty cilia or no cilia at all (madarosis), whereas in our case there were four rows of regular, healthy cilia. According to the table of Fuchs² the normal co-efficient for the age of 16 years is 1.6 while in our case it is 1.24.

Summary

1. A case of congenital shortness of the lids in the vertical direction ("vertical shortness"), combined with tetrastichiasis, is presented.
2. A partial eversion of the cutaneous part of the upper lid margin was observed.
3. In spite of the existing lagophthalmos the eyes remained free from irritation or inflammation, possibly because of the co-existing hypertrichosis.
4. In the sulcus subtarsalis a dark line was apparent due to the hair follicles of the supernumerary cilia showing through the conjunctiva. These have probably taken the place of the Meibomian glands occupying their ducts.

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RESEARCH IN ZURICH

"Summary of the work on the pathological changes in the aqueous-humour and the blood-aqueous barrier, at Zurich."*

BY

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THE research at present being carried out at the University Augenklinik at Zurich is devoted to the study of the aqueous humour and the blood-aqueous barrier.

This work was started by Professor Marc Amsler and his associates in Lausanne in 1940, and was mainly concerned with the albumen and cell-content of the aqueous in disease of the anterior segment. Since the Professor's appointment as Director the work has been continued and augmented at this clinic.

The aqueous humour

The specimen of aqueous humour is obtained in the following way:—A smear and culture are taken from the conjunctival sac. The lids are then held open by a speculum, and a small scraping of corneal epithelium is taken from the lower and outer part of the cornea about three millimetres from the limbus. This area is painted with 5 per cent. iodine solution. The eye is now fixed by an assistant, and a puncture made through the painted area in a plane parallel to the iris. This puncture is made with a special needle devised by Professor Amsler which is placed on a 1 c.c. (tuberculin) syringe.

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The aqueous is then slowly withdrawn, and the needle removed in the same plane as that of its insertion.

This relatively simple procedure can easily be performed in the out-patient department.

The following investigations are made on the specimens obtained.

The conjunctival smear is examined after staining, and the state of the cells noted. The result of the conjunctival culture is correlated with the findings in the culture of the aqueous. The cell scraping of the corneal epithelium is also examined microscopically. A drop of the aqueous is used in the Pandy reaction (this reaction for the quantitative detection of albumen being selected for its reasonably accurate clinical results on small specimens). Another drop is placed in a counting cell (Fuchs-Rosenthal) to give a true figure of the cells/mm³. A culture is taken in a broth medium (Rosenow). The remaining fluid is placed in a capillary centrifuge tube in which the centrifuged matter is deposited on a cover-glass; this specimen is stained with the May-Grunwald and Giemsa reagents and mounted, the slide is then examined to ascertain the type of cell present. If there is sufficient fluid a specimen is also stained by Gram's method.

All this has to be carried out on one-fifth of a c.c. of fluid or less, and the apparatus used is adapted for these minute quantities.

These examinations were controlled by an investigation of 300 normal eyes.

Serum reactions were carried out on a few cases early in the series, but were given up on account of the greater need for the fluid in other investigations. It was found that the W.R. in the aqueous is never positive if the blood reaction is negative, and even with a positive blood reaction unless there is albumen present the result is negative.

So far just over 1900 a.c. punctures have been performed in the course of this research.

Findings

1. In accord with other authors who have studied this question, it has been found that the normal aqueous contains between 0 and 1 cell/mm³.

2. In anterior uveitis and deep ulcers of the cornea, the number of cells is increased in varying proportions. The figures most often obtained vary between 30 and 200 cells/mm³. A few cases rise even to 100,000 cells/mm³.

3. In acute inflammations, the albumens are increased in proportion to the number of cells.

4. In chronic inflammations and other diseases it has been found constantly that there is a dissociation between the albumens and the cells, comparable to that observed in the pathology of the cerebro-spinal fluid.

5. The researches on the morphology and origin of the cells has proved that they are derived from the blood and the neighbouring tissues. The predominant rôle of the reticulo-endothelial system in a great number of cases of chronic irido-cyclitis has been noticed.

6. The phagocytic and antitoxic function of the cells desquamated into the anterior chamber has been indicated by the presence of micro- and macrophages in the specimens.

7. Out of 651 cases of exogenous and endogenous irido-cyclitis, keratitis and panophthalmitis, 19 per cent. showed large reticulo-endothelial cells which had phagocytosed micro-organisms. Those that were most commonly seen were cocci with variable morphology and staining—thought to be staphylococci in most cases. In perforating wounds one may find Morax-Axenfeld bacilli and pseudo-diphtheroids. Tubercle bacilli have never been found, except in one unusual case in which a positive culture was obtained.

Cultures are very rarely positive (about 3 per cent.) even with strongly positive smears. Altogether the cultures have consisted of

7 Staph. albus.	1 Tubercle bacillus
1 Staph. aureus.	1 Morax-Axenfeld bacillus.
3 Pneumococcus.	6 Pseudo-diphtheroids and
1 Streptococcus.	other unidentified organisms.

8. The presence of micro-organisms in the anterior chamber in the course of keratitis and anterior uveitis throws a light on the aetiology in many cases.

9. The absence of tubercle bacilli in the numerous specimens studied causes doubt as to the preponderant rôle which is attributed to them in the pathogenesis of chronic uveitis, especially as other microbes have been found.

10. The phagocytosed micro-organisms which have been found have indicated the possibility of focal infection, and in many cases a focus has been found suggesting a strong aetiological factor.

The blood-aqueous barrier

Following the work of Ehrlich in 1881 on the use of intra-venous fluorescein (when he noticed that the dye found its way into the anterior chamber); this method has been adapted to the needs of the research here on the permeability of the blood-aqueous wall.

The fluorescein is injected intra-venously and relative measurements of its concentration are taken at suitable time intervals during the next 30 minutes.

This is done by means of an adapted slit-lamp in which the illumination of the anterior chamber can be reduced by a resistance while the eye is kept under observation, until the green colouration of the aqueous is no longer detectable. Readings are then taken from an amperemeter in the slit-lamp circuit.

These readings are plotted on a chart in graph form; time/amps resistance over a period of half an hour.

A preliminary investigation was carried out on 200 normals. These were carefully selected from individuals in which there was no evidence of ocular or general disease.

The results in these cases show that:—

1. There is a normal excretion of the dye into the anterior chamber. The analysis of the 200 cases shows that there is a regular and gradual rise of fluorescein concentration during the time of examination.

2. The permeability curves of the two normal eyes of an individual are the same.

3. The day to day variations in a normal eye are well within the limits of the normal band.

4. The normal for children up to about 15-17 years, is a little higher than the normal band for adults.

This research was then directed to the study of the altered permeability in disease, at first only in ocular disease, but more recently in cases with general diseases.

More than 1,000 patients have been examined in this way. The pathological curves of increased permeability are situated above the normal band and are characterised by an immediate rapid rise of concentration to a point where the concentration remains almost unaltered.

The findings in disease are as follows:—

1. Irido-cyclitis. All degrees of permeability increase, from the slightest forms to the most severe are seen, corresponding to the severity of the irido-cyclitis. There is a possibility of early diagnosis in cases where no other sign is visible. A control can be made of therapeutic effect on the permeability.

2. Glaucoma simplex. Some cases show normal or slightly increased permeability, while others show a considerable rise in permeability. It is possible that these alterations in the findings show a differentiation between true simple glaucoma and those tending to an inflammatory aetiology (on these points one's treatment may be altered).

3. Traumata. Contusion and perforating injuries produce a great increase of permeability. It can be seen in cases of unilateral injury, that there is a bilateral rise of permeability although the uninjured eye shows a less marked increase.

4. Other ocular affections causing an increased permeability. As examples: central venous thrombosis, periphlebitis, retinal detachment, choroiditis.

5. General diseases showing an increased permeability (with no sign of ocular inflammation). Bronchial asthma, gastric ulcer, generalised oedema, chronic nephritis, epidemic hepatitis, rheumatic

polyarthritis, obesity, and above all diabetes (40 per cent.), and malignant hypertension. In purpura there is some parallelism between increased permeability and the Rumpel-Leed phenomenon.

6. Pharmacological effects. The pharmacological effects of some drugs commonly used in eye treatment, or which are known to have an effect on the capillary permeability in other parts of the body, have been studied. It has been found that pilocarpine increases the permeability, atropine and homatropine have no effect, adrenalin and l-glucosan diminish the permeability. Sub-conjunctival injections of slightly hypertonic sodium chloride increase the permeability, calcium injected intra-venously in a dose of 10 c.c. of a 20 per cent. solution in many cases has a diminishing effect. Sub-cutaneous histamine in small doses has an enormously increasing effect.

The Tyndall effect

Since this work was taken up at Zurich, further aspects have been enquired into.

The beam of light from a slit-lamp passing through a proteinaceous aqueous in the anterior chamber shows itself in a flare described by Tyndall in 1869, and called the Tyndall effect.

This can be used as a clinical sign in diagnosis and prognosis of infections of the anterior segment. The Tyndall effect is more marked the higher the protein content of the aqueous and this is measured by a Tyndall photometer. This also is a modified slit-lamp in which part of the illumination is reflected through a pair of crossed polarising filters to the microscope. On looking through the eye-piece one sees an oblong strip of light the brightness of which can be adjusted by rotation of one of the filters.

The image of the slit-lamp beam passing through the aqueous is seen immediately above the polarised image. The latter is adjusted until the appearance of both images is identical, and an average of four readings taken.

The results can be plotted on a graph (the Tyndall Nephelogram) giving an accurate record of the variations of the phenomenon.

When plotted against time the graph will show a rise and fall which corresponds closely to the clinical deterioration and improvement, the more useful because of the early appearance of this sign in disease of the anterior segment.

In comparing the uses of the Tyndall effect with that of the fluorescein permeability, we see that the Tyndall effect shows the permeability of the damaged blood-aqueous barrier to proteins and large molecules; while the fluorescein permeability shows the more minute changes in the permeability of the wall to smaller particles. Secondly, whereas the normal barrier will not allow a positive

Tyndall effect, it will allow fluorescein to pass through. Thus the fluorescein will give an earlier and more sensitive impression.

The most recent trends here are aimed at finding out the effect of pathological aqueous on a tissue culture of fibrocytes. It is suspected from clinical observations that the pathological humour will have a detrimental effect on these cells. The work has only just commenced (November, 1946) and no results are available yet.

The aqueous is now also being examined under the ultra-microscope to study the cells, bacteria, and the number and size of the protein particles.

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A GRÖNBLAD-STRANDBERG SYNDROME*

BY

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ANGIOID streaks are a rare form of retinal degeneration and are recognisable by a network of streaks varying in colour from reddish to dark brown. The two components of the syndrome, that is, angioid streaks and pseudo-xanthoma elasticum could be clearly seen in our case. It is suggested that in this disease there is a degeneration of the elastic tissues throughout the body.

* Received for publication, January 18, 1947.

Case Report

Hüseyin K., a worker aged 35 years, complained of blindness in the right eye and was admitted to hospital. He had never before noticed anything strange about his eyes. Personal and past history had no bearing on the case. We questioned him carefully but could not find any trace of a similar eye disease in any relative. Urine analyses showed nothing pathological, arterial blood pressure 70/100 m.m., red and white B.C. number, blood picture and internal organs were normal. Radiological examination of the lungs revealed nothing pathological. Mantoux test and Wassermann reaction were negative, blood cholesterin normal. Externally both eyes were normal. In the right eye central vision was lost, and in the fundus there was a second network behind the retinal vessels, resembling the latter (Fig. 1). Round the papilla was a black, irregular ring from which started many irregular streaks, with ragged borders and of varying width. The streaks were behind the retinal vessels and in front of

RIGHT

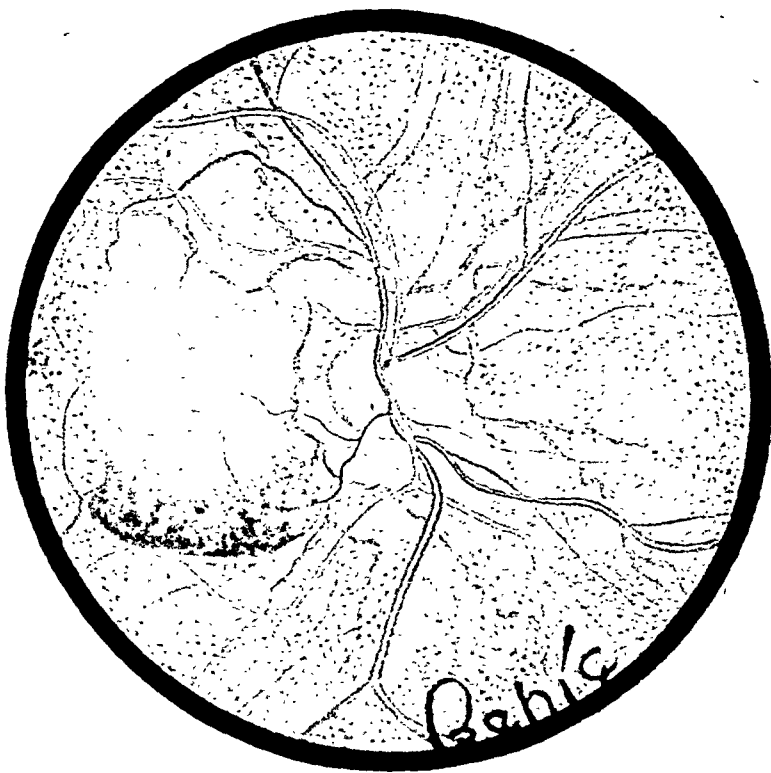


FIG 1.

the choroidal ones. The fundus picture did not reveal whether they belonged to the retina or choroid. Although the streaks usually ended before reaching the peripheral parts of the fundus, some could be seen to meet it. There was a large yellow lesion in the fundus, extending 1 mm. into the vitreous, filling the macular area and reaching the papilla. The lower part of the lesion was surrounded

LEFT

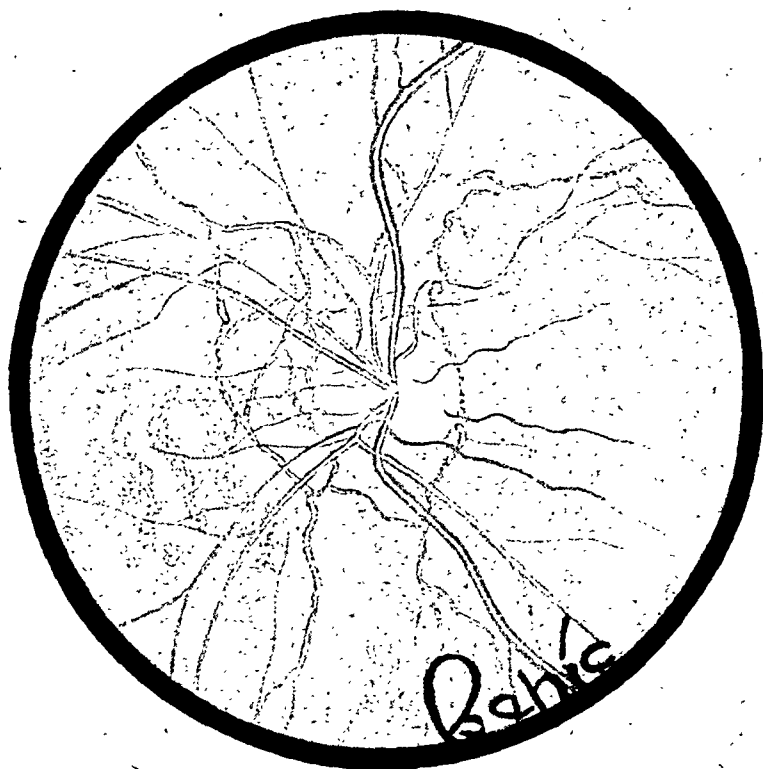


FIG. 2.

by a deep retinal haemorrhage. Near a small artery was a second small haemorrhage, temporal to the macular region. Since the patient had no central vision in the right eye, we obtained its visual field by the Schlosser method. A large central scotoma was revealed (Fig. 3). The left fundus was also abnormal with pigmented streaks as in the right eye, starting from the peripapillary ring and leading to the peripheral parts of the fundus. There were more streaks than in the right eye (Fig. 2). There were three choroidal atrophic patches round the papilla. No macular lesion was present, vision

was 20/25 and visual field normal (Fig. 4). Only macular lesions interfere with vision. Pigmented lines do not apparently affect it.

In 1889 Doyne described the characteristic appearance of the fundus as a network similar to the vessels. Three years later the disease was observed by Plange, Stephenson and Knapp, and named angioid streaks as a clinical entity. The aetiology of the disease is

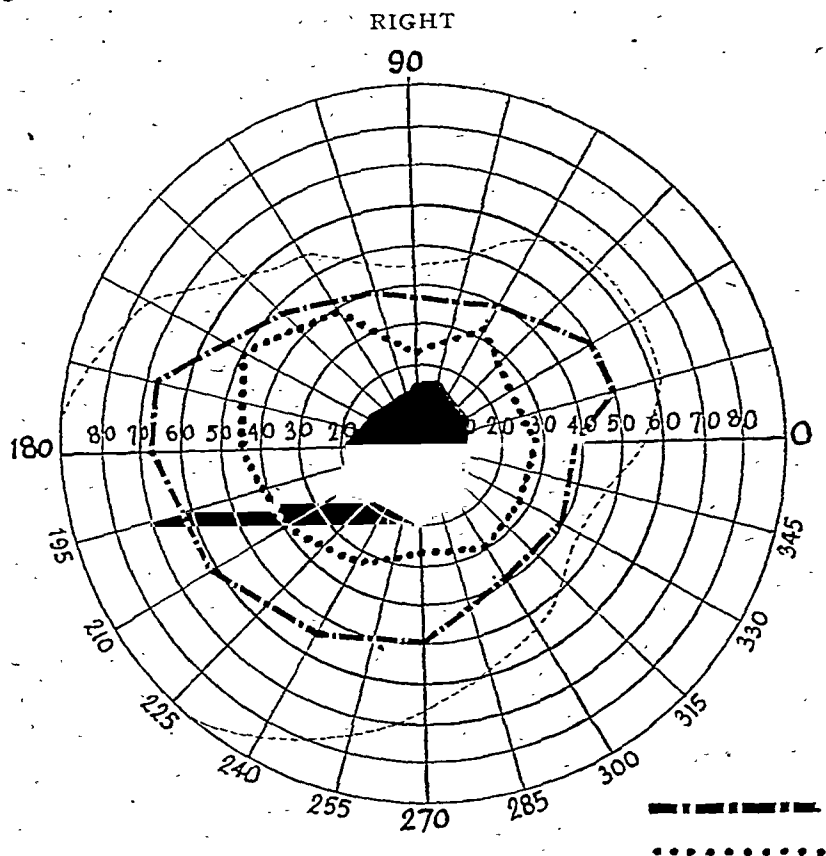


FIG. 3.

not well known. Doyne and Plange suggested that the streaks were haemorrhagic in origin and this was elaborated by Collins. He believed that the haemorrhage was intra-choroidal and that haematogenous pigment was deposited in the perivascular spaces.

The formation of supernumerary new vessels was suggested by Lister and others. Both theories were nevertheless found unsatisfactory.

The deformation of the inner tissues of the eye, particularly of the pigment epithelium, was suggested by Walser, Alt, Coppez, and Danis, the folding being probably produced by some exudative

LEFT

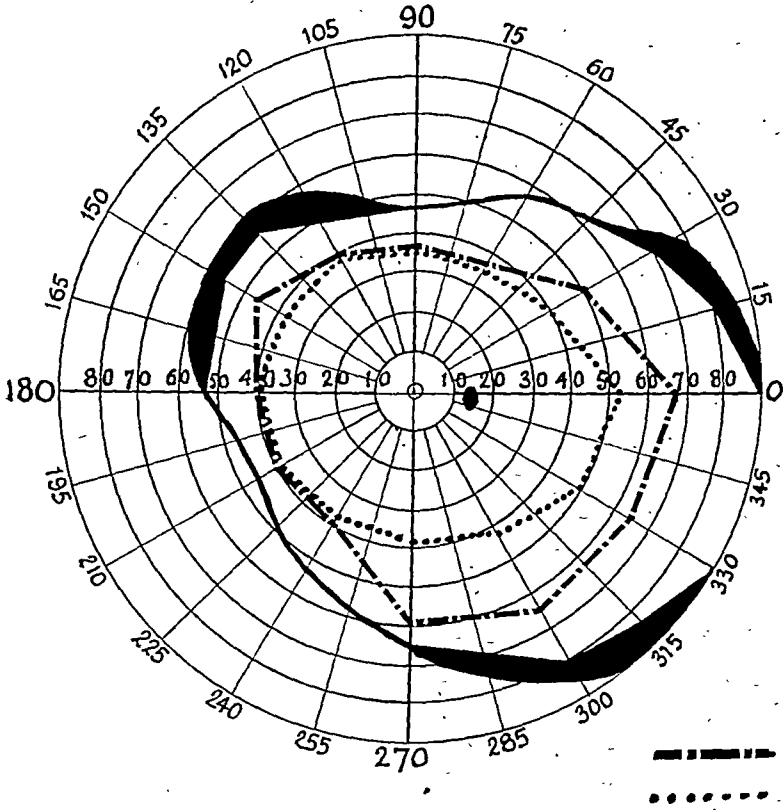


FIG. 4.

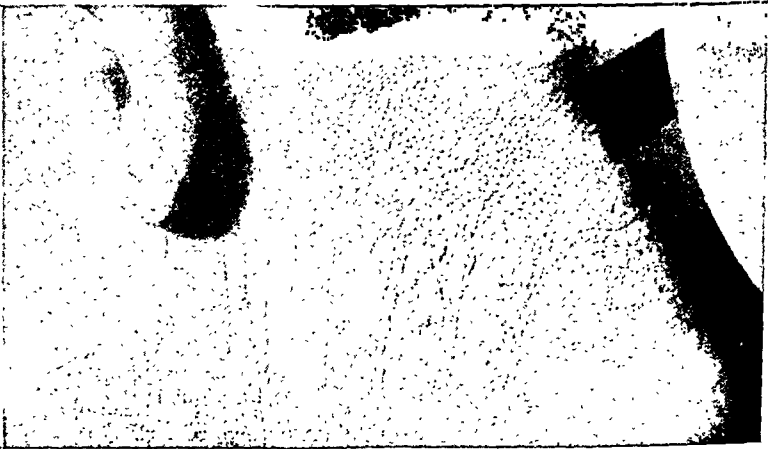


FIG. 5.

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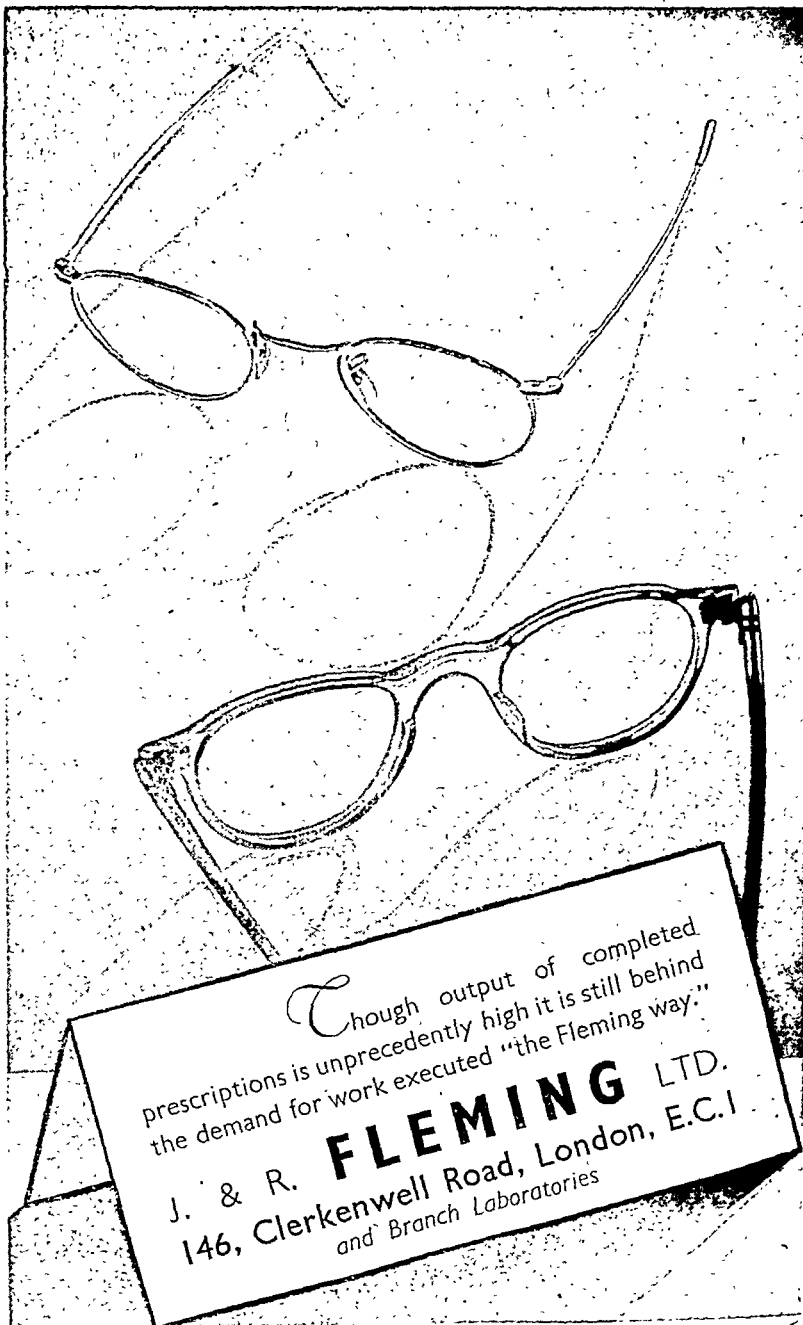


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process in the choroid. Affection of the retina itself by the accumulation under it of pigmentary debris is more fully authenticated by Law's histological findings. This theory, however, was also unsatisfactory.

Deterioration of the elastic elements of Bruch's membrane leading to the formation of fissures was suggested as the aetiological factor by Kofler and Lohmann. The theory attracted little attention, however, until the association of angioid streaks with pseudo-xanthoma elasticum was pointed out by Grönblad.

Our conclusions are in accordance with the last theory. It is a most uncommon disease of obscure aetiology, affecting the elastic tissues throughout the body.

The skin lesions, pointed out by Grönblad, have the appearance of buff or yellow patches situated on the neck, abdomen, flexures and chest. The first two manifestations were apparent in the above-mentioned case. The presence of two conditions together was later observed in many cases. Statistics of Grönblad revealed the fact that 57 out of 67 cases skin and fundus appearances together. The association of two lesions was given the name of Grönblad-Strandberg syndrome.

Angioid streaks are caused by the widespread degeneration of the elastic tissues, and can be observed in the posterior pole and in the lamina vitrea. In the elastic structure of the lamina vitrea and in the inner layers of the choroid one may observe some degenerate patches. These patches appear to be due to the reduced resilience of the elastic tissue and may result in fissures and lines. In our case extensive parts of the skin of the neck and the abdomen had a

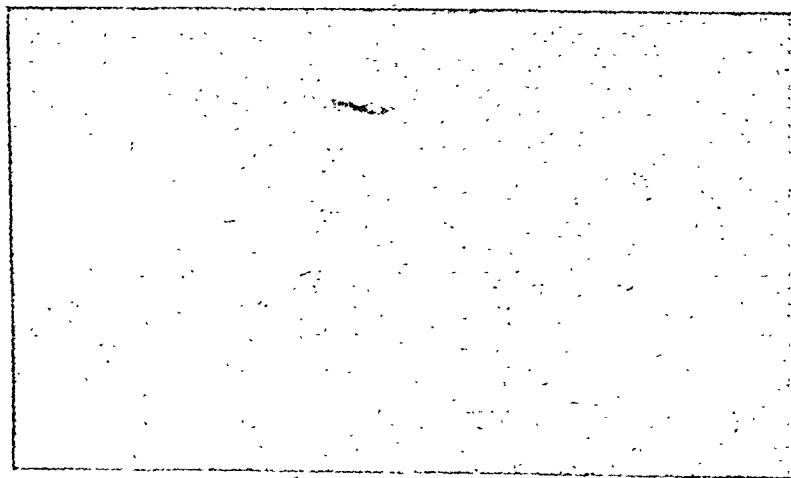


FIG. 6.

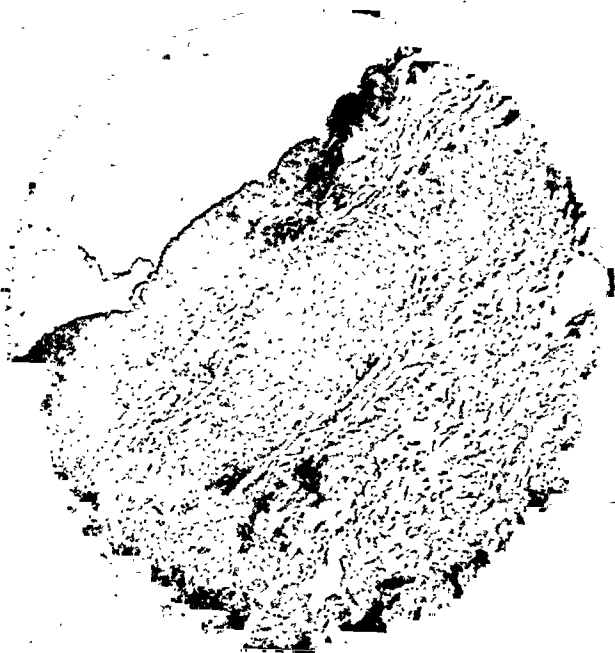


FIG. 7.

parchment-like appearance, and raised yellow streaks and patches could be clearly seen (Figs. 5-6). Although trauma has been quoted as an aetiological factor, no trauma could be noticed in this case.

Pathological examinations were made of the skin of the abdomen:—

“A slight hyperkeratosis may be observed in some parts, and an increase in pigmentation in others. In comparison the epidermis has become thinner, and shows accumulations of large protoplasmic vacuolar cells. The collagen fibres have been broken and diminished in size. Scattered infiltrations of inflammatory cells appear in some parts. “Anatómico-Pathological Laboratory, Gülhane Hospital (Fig. 7).

The above findings confirm the degeneration of the elastic elements of skin. Our case, having characteristic ophthalmoscopic and skin lesions, is a typical example of the so-called Grönblad-Strandberg syndrome.

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 Bd. 114, Nr. 8.

AN IMPROVED DARK-ADAPTOMETER*

BY

WALTER KOCH

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SOME time ago an instrument was described in this Journal intended for testing dark-adaptation in normal and sub-normal patients. However, when it was tried to investigate gross disturbances (e.g., retinitis pigmentosa), the need was felt for a wider range of readings. When re-designing the apparatus provisions were also made to change the colour of the test-light if desired in order to make the instrument more versatile.

As the general outlay of the apparatus has remained the same except for two relevant items, the reader is referred to the previous paper. In the former design (see previous paper, p. 236, drawing I, "9") a blue glass plate was inserted into the path of rays; as this glass plate could not be easily removed, the light of the stimulus was unvarying blue. The same drawing also showed (see "11") how the intensity of the test-light was being reduced by openings of different size drilled into a metal-strip. A limit of this method was given by the fact that a very fine hole is rather difficult to drill, to keep clean, and to center.

In the present design the aforementioned limitations have been overcome as follows:

(a) *Changing the colour of light*: A double-walled, rotating disc (see 9A in this paper) replaces the blue filter (9) of the former design. This disc is similar to that used in ophthalmoscopes for changing lenses, and has six openings. Into five of them filters of different colour can be inserted; the sixth hole remains empty, thus passing uncoloured light when brought into position. In the photo the empty opening (6) is shown centered.

(b) *Altering the intensity of light*: A disc (see 11A in this paper), similar to the colour-filter disc (9A) takes the place of the metal strip (11) used in the previous design. The six openings in this disc are all of equal size and accomodate six grey filters of different transmission. These neutral screens may be obtained from Messrs. Ilford but if less accuracy is needed they can be prepared quite easily by exposing and developing photographic film. Their density* is determined by photometry. As very dark screens can be tested only with

* Received for publication, February 6, 1947.

* Density means $\log_{10} \left(\frac{I_0}{I} \right)$, where I_0 stands for incident light and I for transmitted light; thus a filter transmitting 10 per cent. of the full (100 per cent.) incident light has a density of: $\log 100 - \log 10$, or: $2 - 1 = 1$.

difficulty, several more transparent neutral filters can be combined as to give the required density. As done for the disc carrying the coloured filters, the openings are marked from "1" to "6". In the photo grey screen number three (with a density of 0.40) has been brought into position. The full range of densities is shown in table I:

TABLE I

Number of opening:	"1"	"2"	"3"	"4"	"5"	"6"
Density:	2.48	1.49	0.40	5.78	4.63	3.60

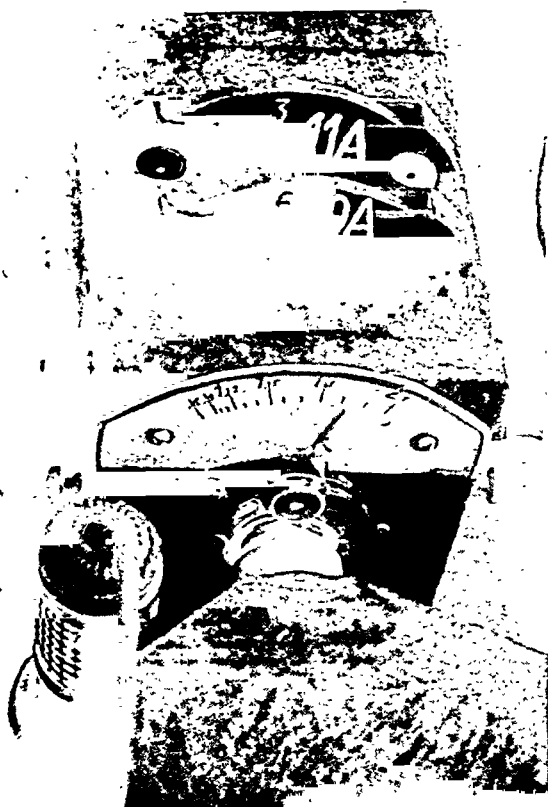
In order to determine the relative densities of our screens, one of them, *e.g.* "1" is assumed to be zero. Thus the density of screen two becomes: $1.49 - 2.48 = -0.99$, the density of screen three: $0.40 - 2.48 = -2.08$ etc. Table II lists these values:

TABLE II

Number of opening:	"1"	"2"	"3"	"4"	"5"	"6"
Density — 2.48:	0	-0.99	-2.08	+3.30	+2.15	+1.12

The calibration proper is performed without colour filter, but with grey screen one in position; a light-source calibrated by the National Physical Laboratory, Teddington, serves as a standard. By stopping down the diaphragm of the standard and of the adaptometer simultaneously, a calibration curve is obtained. Though these figures could be directly entered on the cardboard attached to the face of our shutter, it is more convenient to make a correction for blue light, as this colour is preferably used in dark adaptation tests. In the uncoloured light of our lamp the blue filter itself has a density of 2.37; thus 2.37 has to be deducted from the values obtained in the calibration chart. The corrected figures are now entered on the cardboard. The scale (as shown in the photo) reads directly logarithmic units of millimicrolamberts for blue light, if grey screen one is in position. Shifting over to grey screen two which is by 0.99 units more transparent (see Table II) means *adding* 0.99 to the reading of the diaphragm scale. Similarly, as grey screen six is by 1.12 units more opaque than screen one, it calls for a deduction of 1.12. Using white light instead of blue light, 2.37 has to be added (see above).

It will, however, be noticed that no arithmetic at all is involved when testing final thresholds in normal or slightly subnormal cases if blue light is used. For screen one (relative density zero) does not call for a correction. The neighbouring screen (two) which covers most of the sub-normal cases needs simply adding one (or more exactly 0.99).



Thus the threshold shown in the photo has to be read off as: 1.9 (indicated on the scale of the diaphragm), plus 2.37 ("6" on the colour-filter disc indicating white light), plus 2.08 (due to grey screen "3"), the total value being 6.35 log units of millimicrolamberts.

Summary

A dark-adaptometer is described which covers a wide range of intensities of light stimuli and allows the use of different colours.

REFERENCE

Koch, W.—A new instrument for dark adaptation tests, *Brit. Jl. Ophthal.*, May, 1945, p. 234.



CIRCULATORY STUDIES OF THE FUNDUS OF THE EYE*

BY

P. WEINSTEIN and J. FORGÁCS

BUDAPEST

OUR investigations assumed first that the difference of pressure between the arterial and venous parts of the capillary system acts as circulatory motor, further, that spontaneous venous pulsation of the fundus of the eye is a sign of a pressure prevailing within the central retinal vein lower than the intra-ocular tension. One hundred cases were examined: fifty of them showing spontaneous pulsation, the other fifty without such. Hardly any difference concerning number of hypertonics and hypotonics, ocular tension, age, sex and brachial blood-pressure could be distinguished between the two groups in question (see Table). With those cases in which no

TABLE I

	Pulsating cases 50	Non-pulsating cases 50
Hypertonia	15	15
Hypotonia	7	6
Ocular tension	15-26	13-23
Age	16-79	16-76
Sex	25-25	31-19
	♂ ♀	♂ ♀
Blood-pressure	164/92	160/90
Appearance of arterial pulsation (mm. mercury suction)	97	141
Appearance of venous pulsation (mm. mercury suction)	27	59
Complication:		
Retinal haemorrhage	2	6
Chorio-retinitis	2	4
Papilloedema	1	2
Atrophy of the optic nerve	1	—
Total	6 (12 per cent.)	12 (24 per cent.)

spontaneous venous pulsation was present, such was elicited by applying Kukan's ophthalmo-dynamometer. We did not transpose the mercury millimetre values at the moment pulsation becomes visible, into values of retinal blood pressure, but in the table only

* Received for publication, January 13, 1947.

that mercury millimetre value of suction is represented, at which venous or arterial pulsation becomes first evident.

The difference of the two groups manifests itself in two directions the drop from arterial and venous diastolic pressure established at a higher level with the non-pulsating cases, than with the pulsating ones (see Fig. I), although the value is the same, respectively the difference between arterial and venous pressure is essentially larger with the pulsating eye. The following has to be considered in

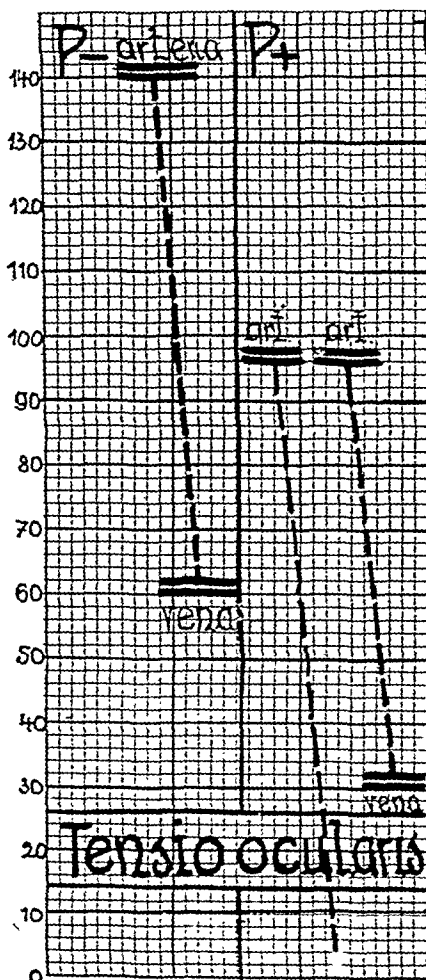


FIG. I.

Suction-pressure exerted on the bulb of the eye by ophthalmodynamometer in mm. of mercury, which elicited the first arterial or venous pulsation respectively. P-: non-pulsating cases, P+: spontaneous pulsating cases.

evaluating these data: Kukan explains his method so that when he exerts suction on the sclera by the disk, the rim of it presses the bulb of the eye in the same ratio, and thereby the intra-ocular pressure is increased in such a degree, that it surpasses the pressure prevalent within the vessels and thus causes pulsation within the same. Lately Dubois and Fischer from Weve's clinic (*Ophthalmologica*, 1941), proved that the intra-ocular pressure is not increased by this suction method, but the stretching of the sclera changes and the pressure of the retinal vessels decreases below the intra-ocular pressure thereby, and then pulsation ensues in them.

The other difference between the two groups is, that with non-pulsating cases the incidence of ophthalmological complications is 24 per cent. while in pulsating cases it is 12 per cent.

The difference between the two groups, namely, that the drop of pressure with pulsating cases is greater between the arterial and venous part of the retinal circulatory system than with the non-pulsating, and that the entire pressure-level of the latter is shifted higher and that incidence of retinal complications is only half with pulsating eyes as with non-pulsating, each follows from the other. The fact that arterial pressure is established at a higher level signifies that an obstacle prevails with the capillaries, to overcome which the pressure of the central retinal artery is increased. If again pressure is higher within the vein, that means worse circulatory conditions than when spontaneous pulsation prevails, because then the drop of pressure between artery and vein is higher and therefore circulation is more continuous. The following cases serve as instances:

1. Plesh curve of very large amplitude, high blood-pressure, spontaneous retinal venous pulsation absent, severe alterations of the fundus, nephrosclerosis.

2. A curve completely similar to the first, similarly increased high blood-pressure, spontaneous pulsation, intact fundus.

3. Curve of large amplitude, high blood-pressure, spontaneous pulsation, fundus haemorrhage and retinitis foci, complete restitution.

The presence of spontaneous venous pulsation with the two latter cases evidently signifies satisfactory capillary circulation and secures retinal supply and disposition for improvement, while with the first case despite high blood-pressure the circulation of the fundus is bad, because the level of the venous pressure is also increased, thereby the terminal points of the pressure-drop converging, the circulation becomes imperfect.

What about hypotonic cases established at a lower level of pressure? As long as the terminal points of the circulatory system show a difference of physiological pressure-drop, which keeps intact circulation going, this latter functions without trouble. But if this drop of pressure decreases, be it by further decrease of arterial pressure or increase of venous, the circulation becomes worse, as with those

hypotonic cases showing spontaneous venous pulsation because this augments drop of pressure. According to Lauber and Sobansky ocular tension is significant with such hypotonic individuals regarding retinal circulation its relative increase already inhibiting retinal blood circulation. Our own investigations have not demonstrated any difference between hypertonics and hypotonics, pulsating and non-pulsating patients regarding ocular tension. According to Nakayima's experiments, persons using steam baths show a decrease of brachial diastolic pressure without exception, on account of the dilatation of the peripheral small vessels, similarly the diastolic pressure of the central retinal artery decreases, so that spontaneous arterial pulsation ensues and in the majority of cases even venous pulsation becomes evident, the system establishing a lower level of pressure, but ocular tension does not change in the course of the whole process. Normal ocular pressure evidently proceeds parallel to sudden increase or decrease of blood pressure, but not in ratio to continuously developing slow alteration of pressure.

Regarding the peculiarities of spontaneous venous pulsation or the artificially elicited one, venous pulsation elicited by pressure is characterized as already described by Fritz, by sudden increase and stop, according to Fritz a sign of satisfactory capillary permeability and suitable flow, or it increases gradually and slowly.

Or pulsation shows a fine vibration during the whole time, a transition towards that group, where no venous pulsation can be elicited because either capillary circulation is bad or diastolic pressure of the veins increased. Elicited diastolic pulsation of the vein appeared always preceding diastolic pulsation of the artery, and it subsided usually near arterial systolic pulsation ceasing, exceptionally near the cessation of diastolic pulsation.

Spontaneous venous pulsation occurs in 40 per cent. on both sides, 28 per cent. on the right and 32 per cent. on the left eye. Its origin is due, according to Serr, to undulations of the ocular tension synchronous with the revolutions of the heart, ocular tension surpassing venous pressure during systole, and causing compression of the vein. This phenomenon was very clearly visible in cases when the central retinal artery pulsated also and the pulsation of the central retinal vein had not yet ceased: the collapse of the vein exactly coincided with the systolic dilatation of the artery. Of course under pathological conditions there is the possibility of a penetrant venous pulse originating. (Aortic ins., Basedow's disease, cranial tumour). In 10 per cent. of the cases spontaneous pulsation disappears if the patient lays down, as a proof that by increase of intracranial pressure it increases within the vein also and thus surpassing intra-ocular pressure the pulsation ceases, as has been described by one of us already, 1938. (Weinstein: *Brit. Jl. Ophthal.*, 1939, p. 396). In 18 per cent. of the cases besides the pulsation synchronous with the

cardiac revolutions, a second separate rhythm of the vein, a formal allorhythmia is discernible, consisting of a ceasing of the rhythmic pulsation for seconds, frequently 4 to 5 then re-appearing again and being replaced by the original rhythm. Then after shorter or longer intervals, a pause again follows. This phenomenon (*pulsus intermittens venosus retinae*, Fig. 2), has not been described in human

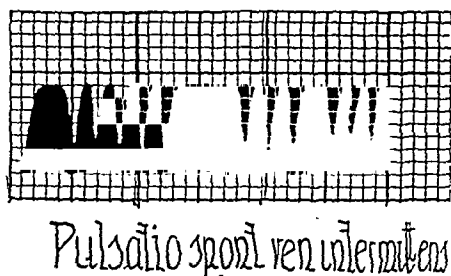


FIG. 2.

Pulsus intermittens spontaneus venosus retinae

ophthalmological physiology and pathology, it has been observed only in dogs as irregularly appearing temporary pulsation and was explained by undulations of tone in the blood-vessels. This intermittent pulsation observed by us, is probably also related to changes of tonicity of the capillaries and is apparent chiefly with vaso-neurotic individuals (migraine, epilepsy). As a practical consideration it follows, that investigating spontaneous venous pulsation the patient should be in a sitting position and the observation should be extended to at least one minute, observing the central retinal vein, because in the prostrate position pulsation frequently it is not visible and often ceases for seconds. With all our cases spontaneous venous pulsation disappeared if the eye was massaged, because then the ocular tension ceased and sank below venous pressure, whereby pulsation ceased for a time. (Seidel and Serr).

Summary

Individuals showing spontaneous venous pulsation have a higher drop of pressure between arterial and venous systems of retinal circulation compared with persons without pulsation, and consequently retinal circulation of the former is more satisfactory.

The level of arterial and venous diastolic pressure values of the fundus of individuals showing no spontaneous venous pulsation are established at a higher level than those of the pulsating group, a circumstance pointing to a circulation inhibited to a high degree within the capillaries.

Retinal complications show an incidence twice as high with cases without pulsation as with those pulsating.

Spontaneous venous pulsation disappears in 10 per cent. of the cases if the patient is laying down, and is of intermittent character in 18 per cent.

Spontaneous venous pulsation disappears if the bulb of the eye is massaged.

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XV. Cong. Ophthal. 1937. Egypte. L'Hypertension arterielle de la retine.

ANNOTATIONS

Moorfields, Westminster and Central Eye Hospital and The Ophthalmic Institute (University of London)

The amalgamation of these three hospitals marks the beginning of a new era in the development of hospital services for eye disease in London. Implicit in this development lies the certainty of greatly improving the teaching and training of ophthalmologists and also the possibility of intensive research into the many unsolved problems of ophthalmology.

More than twenty years ago a scheme for the amalgamation of Moorfields and the Central Eye Hospital was discussed. Some of the preliminary work was done but the scheme failed because it was not thought possible to raise the necessarily large amount of money that would have been required to construct an entirely new hospital with the facilities and equipment then envisaged. This was all the more regrettable as at that time a vacant site adjacent to the Central was available which would have been amply large enough for the projected hospital and still leave space over for such development as the future might require.

Once this scheme was abandoned Moorfields proceeded to reconstruct itself on the very cramped site and the Central—then in a completely new building—just carried on. But in reality neither hospital was quite happy. Moorfields remained cramped and the Central soon found that its new building was not adequate to accommodate the new types of apparatus and the new departments which the developments of the science and practice of ophthalmology made necessary. One has only to mention such things as the slit-lamp, orthoptics, light and X-ray treatment to show what expansion was necessary. A further problem was the training of ophthalmologists. Post-graduate teaching of ophthalmology became an

urgent matter with the institution of the D.O.M.S. and the necessity to accommodate a large number of students added to the congestion. That was the position up to the beginning of the war of 1939/45. During the war it was realised that extensive changes would have to be made if post-war requirements were to be adequately met. In addition to all this it had long been felt that there should be a research institute for ophthalmology with all the requisite laboratories and equipment.

The termination of the war brought a great increase in the number of students and with the conditions prevailing on the continent of Europe it is to be anticipated that their number may increase still further, while the much reduced working hours of the nursing staff added to the difficulties in running the hospitals.

Then came the new Health Act. The Government found itself faced with an inadequate number of ophthalmologists to provide the services for which it had made itself responsible. The result of all this was that Moorfields and the Central agreed to amalgamate and to pool their resources and the University of London undertook the establishment of a teaching institute as part of its general Post-graduate work.

At this stage the Royal Westminster Ophthalmic Hospital decided to join the new amalgamation and in pursuance of these objectives an Act of Parliament was obtained which became operative on January 1st, 1947, establishing the new hospital under the somewhat clumsy appellation which heads this note.

At the present moment the intention is to transfer the hospital services—in-patient and out-patient—of the Central to the other two branches of the combined hospital and to transfer the main laboratory and teaching facilities (pathology, bacteriology, lecture rooms, museums, libraries, etc.), from the other two to the Central which will become one of the constituent institutes of the Post-graduate Federation of the University of London. On the one hand this will centralize the routine clinical work in two buildings instead of three without the loss of beds or clinical facilities, and on the other it will free the building of the Central for systematic teaching and research. In this way it is hoped to improve the post-graduate teaching of ophthalmology in London and to start a central research institute which it is hoped may be not without influence on the progress and development of our subject. Nor is it the intention that development stops at this stage. Neither Moorfields, nor the Westminster is an ideal building for the modern practice of ophthalmology; the Central is not structurally designed for an institute of teaching and research. Moreover two separate hospitals, although intimately associated, and a research institute geographically divorced from them cannot be considered adequate as a permanent policy. The present arrangement is therefore accepted as the best that can be

accomplished in the immediate future; but the long-term plan when facilities are available is to build a new eye hospital and institute in the University area in close association with University College Hospital—which it is hoped will serve as a general hospital—and University College with its Department of Medical Sciences and its scientific laboratories.

On Eye Shades

We confess that we have always classed the eye shade and the eye bath as household domestic appliances. We would not wish to imply that either is *the* domestic remedy, for in Victorian times this term nearly always denoted the brandy bottle; and we have known the case of a rigid and semi-fainting teetotaler immediately recover when it was suggested in all kindness that she should have “a little drop of brandy.”

But if an eye has to be irrigated, an undine, or if none be available, a small tea-pot, is a much better appliance to use than an eye bath; so in most instances we consider a pair of goggles or a pad and bandage to be preferred to an eye shade.

The simplest form of the latter is a D-shaped piece of stiff paper provided with string or tapes to fix round the head. The flesh coloured variety, made of celluloid, one buys at the chemist, deceives nobody and is a potential source of danger. We recall the case of an elderly gentleman who was wearing one and stopped to light a cigarette in the street. His shade caught fire and a nasty superficial burn was the result. Those shades made of black plaited straw seem to us safer and on the whole less unsightly.

There is one state of affairs when an eye shade is a most useful adjunct and that is in diplopia. We cannot speak from personal experience in this instance for we have never suffered from this disability, but if we ever do we shall make a shade out of the best material available at short notice.

When a patient wearing a shade comes up to the desk at which you are seeing out-patients there is no knowing what it may conceal. Anything, from a sty to a severe burn or wound of the eye-ball may be hidden by it, or even an empty socket. It is surprising how often, when the shade is removed, the condition it has concealed from view turns out to be some extremely mild affair, such as a sty. But for ourselves we have to admit that we always experienced a queer feeling of doubt (or shall we say apprehension?) that we never experienced with an open eye, however inflamed it might be. The dread of the unknown is always in excess of the reality.

BOOK NOTICES

An Introduction to the Prescribing and Fitting Contact Lenses.

By FRANK DICKINSON, F.B.O.A. (Hons.) F.A.A.O., and K. G. CLIFFORD HALL, F.S.M.C., F.A.A.O. Published on December 30, 1946, at 2 guineas net.

Contact Lenses are much in the public eye at present, and, in a fair proportion of cases, also in their eyes—though for varying, and sometimes rather short periods of time. The appearance of this book is, therefore, to be welcomed, particularly as the authors, though not medically qualified, have had a large experience in fitting these lenses. In such work, nothing can replace actual experience, and it is stated in the preface that the authors are attempting not so much to teach contact lens fitting, as to outline the principles upon which the practical technique is founded. In the same way, as one cannot learn, say anatomy without dissection, so one cannot learn contact lens fitting without actually doing it—but a reference book is undeniably useful by providing guidance, without which progress may become lamentably slow. The guidance provided can best be appreciated by a list of the chapters, which deal successively with the history of contact lenses, their sphere of utility, the types in use and their optics. Then come chapters on the preliminary investigation, theoretical, chemical and psychological fit, with a final chapter on the ethics of practice and the future of contact lenses. There are appendices dealing with contact lens trial sets, extracts from case records, and contact lens terms and abbreviations.

The book is admirably illustrated, with numbers of photographs which show clearly the various techniques for inserting and removing contact lenses, and also some coloured illustrations of the fluorescein method for determining fit. In this connection the authors emphasise the importance of corneal clearance—i.e., of avoiding contact between the back of the lens and the front of the cornea—and of the avoidance of bubbles. They also give details of the various solutions to be used for filling the lenses, prior to insertion. If the new type of lens with a ventilating hole is used, however, all this is changed, because the lens is put in dry, there is capillary contact between it and the cornea and the presence of bubbles is a definite advantage in preventing Sattler's veil. It is not fair to criticise the authors on these counts, however, because the paper describing the new lens is too recent for inclusion in their book, which, in the opinion of the reviewer, is an excellent *ex parte* statement of the uses and properties of various types of contact lenses. Its detailed perusal can certainly be recommended to any who entertain the idea of fitting contact lenses, and wish to know the scope and indications for these appliances. The actual fitting will probably always remain an art, rather than a

science, in the same way as sculpture or painting, but, inasmuch as the sculptor or portrait painter is helped in his work by a knowledge of anatomy, so is the contact lens fitter helped by a knowledge of the scientific background of his art. This knowledge is supplied in full measure by the volume under review.

Principles of the Contact Lens. By H. TREISSMAN, M.B., B.S., F.R.C.S., D.O.M.S., and E. A. PLAICE. Price, 10/6 net.

This small volume is designed as a guide to ophthalmologists in the prescribing of contact lenses, and technical details of fitting are omitted. A concise account is given of those optical principles which apply to contact lenses, so that the advantages of these appliances can be fully appreciated. The indications are listed, and the various types of lenses described. A description is given of the preliminary test which should be carried out by the ophthalmologist, and the book ends with some discussion on tolerance. It is well arranged and clearly written, and will be very useful to ophthalmic students, while many practising ophthalmologists will read it with profit.

Report on Defective Colour Vision in Industry. By a Committee of the Colour Group of the Physical Society. Taylor and Francis, Red Lion Court, Fleet Street, E.C.4. Price, 3/6.

Colour vision tests have long been compulsory for occupations in which defective colour vision might prove positively dangerous to life; e.g., the Royal Navy, the Mercantile Marine, and railways. It has been well known, of course, that there are many occupations which cannot be carried out efficiently by colour-defective people. There has, however, been no organized effort to prevent such people from entering these occupations, and such effort could indeed only have been organized after a preliminary investigation of the problem. This has now been excellently done by a committee of the Colour Group of the Physical Society. The presence of Messrs. J. H. Shaxby, W. D. Wright, and F. H. G. Pitt on this Committee is an assurance that the scientific aspects of Colour Deficiency were adequately represented.

The report gives first a brief and accurate account of the nature and incidence of Defective Colour Vision. This is followed by a very useful description (with bibliography) of practically all the colour vision tests in current use. The concluding sentence of this section conveys a salutary warning: "It may be stated that no one test, other than the very expensive colorimetric apparatus is capable of detecting defective colour vision with absolute certainty, but that this certainty may be more nearly approached if more than one type of test is used."

The third chapter records the results from exhaustive enquiry

from trade associations and individual firms upon the extent to which deficiencies affected the work of employees. Many of the industries involved are self-evident; *e.g.*, textiles (dyeing and matching), drapery, lithography and photo-engraving, paintmaking, and so on. Others, less obvious, are equally important, *e.g.*, colour-coding of wires and resistances in electrical industries, heterochromatic photometry—likely to be still more important with the introduction of fluorescent lamps, analytical chemistry and the determination of the end points of titrations and the use of indicators, and so on.

Two factors account for the apparent apathy of industrialists in this matter. In the first place many of the workers are women, and colour deficiency is relatively very rare among women. The second is that in most of the trades a colour defective employee can easily be transferred to some other job.

The remedies recommended by the Committee are school and pre-vocational testing of colour vision.

They recommend that "all children should be tested at school at the age of 13 or over, using one of the approved Confusion Chart Tests." They do not shirk the fact that this is a large order. The evidence of Dr. Gale of the Board of Education, shows how difficult it would be for anything like an adequate test of colour vision to be included in the present system of medical examination. Any single test, as the Committee itself states, is an insufficient criterion, and as much harm as good might well be done by labelling pupils as normal, only to find that they subsequently fail for the Navy or Mercantile Marine. Any group test is likely to prove fallacious. The Collins-Drevor Test with Bradley papers has been used, and "nearly 78 per cent. of boys and over 94 per cent. of girls who failed in the examination quoted proved to have no colour deficiency when tested individually by the Ishihara Charts.

Pre-vocational tests are far more practicable and are to be strongly recommended. Their value has indeed been proved in Edinburgh and Glasgow. The Committee say: "In view of the very general acceptance of confusion chart tests, and because they are in fact suitable for a great variety of industries and trades, we recommend their use as a good all-purpose test. But we also recommend that they should be supplemented by a trade test designed for the particular kind of task which will be undertaken under normal working conditions."

The Science of Seeing. By IDA MANN and ANTOINETTE PIRIE.
Pp. 220, with 8 illustrations. Pelican Books (A.157). 1946.
Price, 1/-.

As "Madam How and Lady Why" was written by Charles Kingsley as a book of first lessons in earth lore for children, so the present booklet may be likened to a series of chapters on eye lore

for those who have not been initiated into ophthalmology. It is extraordinary how little even educated people know about their eyes: many do not even know the difference between an oculist and an optician. There was a need for a book of this sort, in which the writing is easy and the style at times colloquial, addressed to the great mass of the population.

Certain widely spread erroneous ideas about eye work being harmful to the eye, glasses tending to weaken the sight and that it is possible to take the eye out, scrape it and put it back again are demolished, and the reader is conducted up the zoological staircase from the reaction of the amoeba to light to vision in the human species.

In discussing myopia the authors say that we know of no means of checking the growth of the child, but if they will refer to Nicholas Nickleby they will find that Mr. and Mrs. Crummles put the infant phenomenon on to an unlimited allowance of gin and water from infancy in order to keep her small. Perhaps this idea needs to be demolished with the others.

Many interesting facts emerge from these pages. Bulls are colour blind and it is not the red cloak that irritates them, but more probably the movement. Bees do not see red; wasps are not mentioned, but the small urchin who pokes a stick into a wasp's nest might disagree as far as wasps are concerned. Seals are highly astigmatic. We congratulate the authors most heartily. The chapters are all good, especially those on colour vision; vitamins and vision; things that may go wrong with the mechanism of seeing; things which may affect the function of the eyes, apart from disease and injury; what the blind can do and how they can be taught.

All those of Victorian status will appreciate the reference to the use of a stereoscope on Sunday afternoons combining amusement with latent instruction.

With regard to the limerick on page 80 we do not claim to be a Hampshire Hog, but would suggest that the village of Cosham in that county would rhyme better with "wash 'em" than East Bosham. We were under the impression that the latter was pronounced "Boseham".

CORRESPONDENCE

MYOPIA AND PSEUDO-MYOPIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—I desire to support Mr. Lavery in what he has said in your last issue on "Myopia and Pseudo-myopia" by Mr. Spencer Walker and I should like to go a step further,

His letter has particularly brought home to me the case of a young man who came to me the other day complaining that he had been rejected in the visual examination for the Master Mariner's Certificate. When he first consulted me a few years ago he was accompanied by his father. I explained to him that though his son would probably pass his entrance examination I was afraid that he would not pass his final because his myopia would most likely have increased by that time.

Now my point is why should such a healthy young man with excellent colour vision and with only a slight amount of myopia, be lost to the services of the Mercantile Marine or even to the Navy, but be acceptable to the land forces. It seems to be perfectly correct for an Admiral or a Sea Captain to view a ship in the offing by means of a pair of binoculars but incorrect for him to do the same thing with a pair of glasses poised on his nose.

Our Faculty of Ophthalmology should endeavour to educate the powers that be to a more sane and reasonable outlook on these matters. Many a worthy Son of the Sea whose ancestors have played their part in making England great has been cast aside as worthless because he had half a dioptré of myopia.

I think the whole subject of myopia merits careful and serious consideration. Is it wise for the M.O.H. to insist on the refractionist getting 6/6 or 6/5 vision with minus glasses or would the pupil have been better treated if his 6/9 vision had been untouched and plus glasses with base in prisms been prescribed to assist his accommodation and convergence?

Yours faithfully,

FRANCIS E. PRESTON

44, QUEEN ANN STREET, W.1
25th February, 1947

REMOVAL OF THE WRONG EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—It was with great interest that I read, in the January issue of the Journal, the interesting article by Dr. H. M. Traquair, concerning the removal of the wrong eye. May I offer a safe and simple method to the profession to avoid this mistake, and may I be allowed to correct a linguistic misinterpretation in this article?

The misinterpretation was due to the translation of the "Soll" in Elschmig's quotation. The "Soll" is equivalent to the Latin "Dicitur" and does not mean that this case has actually occurred; it means that it was said (by someone) that the case has (somewhere)

actually occurred. Thus, Elschnig himself did not experience this mishap neither did it occur in his department—at least not between 1912 and 1932, to my knowledge.

My suggestion is to cut the lashes of every patient's eye to be operated on, the day before the operation, or even two days previous to the operation. This can be done in the ward or in the surgeon's office where all records are easily available and when—in the worst case—the patient may remind the assistant that he is preparing the wrong eye. In Elschnig's clinic, we used to cut the cilia in the preparation room adjacent to the operating room; for the last years, I have been cutting the lashes one or two days before surgery, in all intra-ocular operations. The same procedure could be adopted for the enucleation and will prevent the search for, and possible mistake of, the eye to be operated on.

With sincerest congratulations for the improved appearance of the Journal.

I remain, very truly yours,

K. W. ASCHER, M.D.

2508, AUBURN AVENUE,

CINCINNATI 19, OHIO,

February 28, 1947.

OBITUARY

SIR ARNOLD LAWSON, K.B.E.

SIR ARNOLD LAWSON was the fourth of seven sons of George Lawson (1831-1903). George Lawson was a pupil of Sir William Bowman at King's College Hospital, and went to the Crimea as an Assistant Surgeon in 1854. In May, 1855, he had a very severe attack of typhus fever that produced a complete paraplegia, and he was invalided out of the Army in January, 1856. He was already an Assistant Surgeon at Middlesex Hospital where he was a colleague of John Whitaker Hulke. George Lawson was elected Assistant Surgeon to Moorfields Eye Hospital in 1862 on the same day as Sir Jonathan Hutchinson.

He became Surgeon-Oculist to Queen Victoria, holding this appointment until her death in 1901.

He was a man of great charm, a Tractarian, deeply religious, whose generosity, especially to his hospital patients, has become legendary. To follow such a distinguished father is a heavy task, and this Sir Arnold did in detail except that he confined the professional side of his life to ophthalmology.

He was born at 12, Harley Street (into which his father had moved in 1863) on December 4, 1867, and he was destined to spend his whole life there. He was educated at the Merchant Taylors School and in 1886 entered the Medical School at Middlesex Hospital as Senior Entrance Scholar. He did well as a student, winning the Hetley Prize in 1890 and was Senior Broderip Scholar in 1891, in which year he qualified. He graduated as M.D. of Brussels in 1891 and obtained his F.R.C.S. diploma in 1893.

Sir Arnold soon decided to be an ophthalmic surgeon and joined his father in practice. He was elected ophthalmic surgeon to the Paddington Green Children's Hospital in 1896 after having worked with Sir John Tweedy at Moorfields. In 1900, when it was decided to increase the visiting staff at Moorfields from nine to twelve, he was elected Assistant Surgeon together with John Herbert Fisher and Percy Flemming. He became full surgeon in 1907 and retired in 1914, becoming Consulting Surgeon in 1923. In 1910 he joined William Lang at Middlesex as Assistant Surgeon, succeeding him as full surgeon in 1914. In 1932 he was elected Consulting Surgeon. He served as ophthalmic surgeon to the Hospital of St. John and St. Elizabeth, and from 1914 to 1919 was ophthalmic surgeon to King Edward VII Hospital for Officers. For many years up to the time of his death he was Consulting Ophthalmic Surgeon to the Royal Hospital for Incurables at Putney and to the Royal Medical Benevolent College, Epsom.

In 1914 began his important work in connection with the founding of St. Dunstan's. He became principal ophthalmic adviser to Sir Arthur Pearson in this connection and remained Chairman of the Ophthalmic Advisory Committee until his death. For this work he was appointed K.B.E. in 1920.

In 1940 he was appointed Ophthalmic Consultant to the Navy. Of this he was very proud, refusing to take any fee for his services, saying that it was his contribution to the branch of the Forces in which his son served.

He became suddenly ill in 1918 and the nature of the complaint was not diagnosed. He recovered almost as suddenly but in July, 1921, the true nature of his ill-health became apparent when he had a serious breakdown and he spent six months in a sanatorium. After his return to work he gave up many of his appointments and for five years attended no meetings where many people were congregated. He seemed to have recovered all his vigour so as to lead a busy life to the time of his death on January 19, 1947. He actually saw patients on December 19.

Sir Arnold became a member of the Ophthalmological Society in 1896 and after serving as a Vice-President became Treasurer in 1919, retaining the office until 1947 (when he was elected honorary



SIR ARNOLD LAWSON—1867-1947

member), so that his resignation and death were reported at the same meeting of the Council. In this post he was very successful and the work interested him. He always said he hoped to hold the post for thirty years.

He became president of the Royal Medical Benevolent Fund on the death of Sir Thomas Barlow, and in this position proved a great success. His appeals, more especially the one at Christmas produced much greater funds than previously. He collected large sums for the completion and decoration of the Chapel at Middlesex Hospital in which his father and family had always taken a generous interest.

Lawson's literary output was not large when one considers the length of time he was in practice. He edited and largely re-wrote his father's text-book on Diseases and Injuries of the Eye, in 1903. He published a report on the War blind at St. Dunstan's in 1920; and wrote articles in Latham and English's System of Treatment. He published with Sir James Mackenzie Davidson a monograph on Treatment of the Eye by Radium.

He attended meetings of the Ophthalmological Society and the Ophthalmic Section of the Royal Society of Medicine, which he served as president from 1924 to 1926, taking part in discussions and occasionally reading papers.

Lawson will be much missed, especially by his older colleagues. He was a well known figure to all, as he appeared every year for so long a time at the annual meetings of the Ophthalmological Society to present the treasurer's report.

The funeral took place at All Saints Church, Margaret Street, in the presence of a large representative congregation. He had been connected with All Saints from his earliest years and claimed to have been one of the oldest members of the congregation. He had displayed as a boy his love of music, and especially of church music. He was a regular member of the congregation at the Sunday early services.

He married in 1904, Helen, daughter of Andrew Clark, F.R.C.S., of 71, Harley Street, who was a surgeon to the Middlesex Hospital. She died in 1944 and they are survived by two sons and a daughter.

DEWAN BAHADUR DR. K. KOMAN NAYAR

WE regret to announce the death of Dewan Bahadur Dr. K. Koman Nayar, D.O.M.S. at his home in Madras, on November 1, 1946. Dr. Koman Nayar was one of the leading ophthalmologists in India. He served at the Government Ophthalmic Hospital, Madras, in various capacities from 1912 to 1946 when he retired. He became assistant superintendent in December, 1918, and frequently acted

as superintendent, until in 1938 he was confirmed in that post, and appointed Professor of Ophthalmology, Medical College, Madras. His courteous unassuming manner and devotion to duty were combined with a professional ability and sound judgment appreciated by all who came in contact with him.

NOTES

Hunterian Lecture MR. J. MINTON'S Hunterian Lecture on "Occupational Eye Diseases and Injuries" will be given at the Royal College of Surgeons on June 12, 1947, at 5 p.m.

* * * *

Oxford
Ophthalmological
Congress THE 33rd Annual Meeting will be held at Oxford on July 3, 4 and 5, 1947, in the Department of Human Anatomy. The programme includes a discussion on "The Contracted Socket," to be opened by Professor T. Pomfret Kilner and Mr. H. B. Stallard. The Doyne Memorial Lecture will be given by Professor L. S. Stone, of Yale University. His subject is the "Return of Vision and Functional Polarisation in the Retinae of Transplanted Eyes." Arrangements are being made for a museum of exhibits.

* * * *

Irish Ophthalmological Society THE next General Meeting of the Irish Ophthalmological Society will be held in Dublin on May 22 and 23. Prof. Bernard Samuels of New York will deliver the Montgomery Lecture on May 22.

* * * *

The Illuminating Engineering Society LIGHTING IN COAL MINES, by J. Ivon Graham, M.A., M.Sc., M.I.Min.E. (Summary of a Paper to be given at a Meeting of the Illuminating Engineering Society to take place at the E.L.M.A. Lighting Service Bureau, 2, Savoy Hill, London, W.C.2, at 6 p.m. on Tuesday, March 11, 1947).

INTRODUCTION. . The paper opens with a reference to the report issued in 1944 by the Technical Advisory Committee appointed by the Minister of Fuel and Power under the Chairmanship of Sir Charles Reid which stated that "no man can work properly without good light, and this especially so when conditions are difficult."

The amount of light given by early forms of safety lamp was very low and it is only the capacity of the human eye to adapt itself to low illuminations that enables the miner to do any appreciable work. Mention is made of attempts to improve lighting conditions in mines over a number of years. Although the installation of mains electric lighting or of pneumatic electric lamps has not shown anything definite as regards increased output it has led to a reduction in accidents.

PORTABLE AND CAP LAMPS. Two electric hand lamps were in use prior to 1913 but it was not until 1934 that there was any real improvement in the illumination given by hand lamps. The problem of glare then arose and the soft yellow light and the reduced glare of the improved flame lamps giving 4 candle power was often preferred to the use of electric lamps.

Tests carried out on the maximum candle power given by oil and electric lamps both before and after the shift are mentioned. Suggestions are made by which the best possible utilisation of energy from the battery of electric lamps might be obtained.

Cap lamps giving a higher illumination have become more popular during the last few years and have the additional advantage of reduced weight and freedom of movement of the hands.

The Reid Committee recommended an illumination standard of the order of 0.4 ft. candles in the general working area though it was appreciated that such a standard was impossible with either the hand or cap lamps. Even if, with improvements in the candle power/weight ratio, the illumination given by cap lamps may give the necessary local illumination, the absence of general lighting would still be a drawback.

ILLUMINATION BY MEANS OTHER THAN PORTABLE LAMPS.

(a) *Transportable Self-contained Battery Lamps.* Lamps of this category were the logical development of the miner's hand lamp. The advantages are that they can be readily moved to where required and that illumination of the required standard can be provided on the work. On the other hand there is the nuisance of daily charging, difficulty in obtaining a flame-proof certificate, transport to and from and need of care at the working face and finally the glare produced.

(b) *Mains Lighting.* Mains lighting is now permitted subject to special regulations. A number of trial installations and tests which have been carried out are described. Mains lighting in the Ruhr coalfield, where it is reported the illumination in mines is of a high standard, is also discussed.

LAMPS FOR MAINS LIGHTING. The characteristics of the various types of lamps, e.g., tungsten, mercury, sodium, etc., are

mentioned. Fluorescent tube lighting has distinct advantages including the psychological effect of daylight, high efficiency and low brightness, and the application of this system of lighting is now being developed through a Committee of the Ministry of Fuel and Power and with the assistance of the National Coal Board.

THE PNEUMATIC-ELECTRIC LAMP. Conditions in many pits may not permit the application of mains lighting even with relaxation of the present regulations. Such pits, if equipped with compressed air, have in the pneumatic-electric lamp a means ready at hand for greatly increasing the illumination at the coal face. This lamp is described in some detail together with the cost of operation. The application of vapour-discharge sources and the fluorescent tube to this lamp is also described.

Corrigenda UNFORTUNATELY authors' proof corrections of the paper in our last issue by Professors L. C. Martin and R. W. B. Pearse had not been received when the number went to press. By an oversight the horizontal scale numbers were omitted in Figs. 3 and 4. The entire logarithm scale shown in the squares starts at 2.5 and ends at 1.0 reading from left to right. In addition the following errors should be corrected: p. 131, last line but one, The acuity object (Fig. 1) should read The acuity object (S, Fig. 1). P. 135, Table III, degree signs should be added in each first compartment of the six columns as follows; $3^{\circ}/61^{\circ}$ and so on. Page 137, Filters. The 'm' in Ilford minus Green should have been a capital. In No. 8, delete (γ): in No. 10 the last half of the parenthesis should be added. Page 138, Table IV. In Ilford Minus Green column; the 18th figure should read 3.06, and in the same table, 2nd line, last column, 0.772 should read 0.77. Page 142, Table VI, 1st line, last column, 0.066 White should read 0.063. Page 144, the final word of the first paragraph should read tests not texts. At the end of the paper the following sentence should be added. "Our thanks are due to many observers who took part in the trials; and to the Director of Research, Admiralty, for permission to publish this report."

IN the paper on "Posterior ring abscess of metastatic origin in Behcet's Disease" (by A. Feigenbaum and W. Kornblueth), December, 1946, p. 729, para. 1, line 9, for "general" read "genital."

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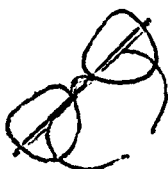
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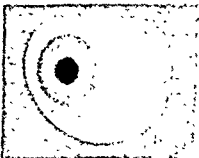
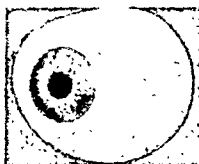
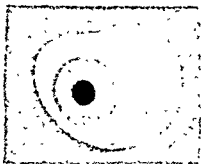
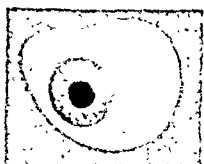
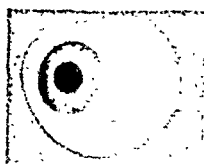
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THE BRITISH JOURNAL OF OPHTHALMOLOGY

MAY, 1947

THE TROCHLEAR NERVE IN THE HUMAN EMBRYO AND FŒTUS

BY

E. R. A. COOPER

DEPARTMENT OF ANATOMY, UNIVERSITY
OF MANCHESTER

IN a recent paper (Cooper, 1946 (a)) the development of the oculomotor and trochlear nuclei was described, and certain details of the origin and course of the oculomotor nerve were given. Since the trochlear nerve pursues an anomalous route to a decussation on the dorsum of the brain stem, further study was necessary before an adequate account could be submitted.

There are three questions relevant to the problem of the trochlear nerve:

1. Why is there a decussation?
2. Why is the decussation on the dorsal aspect of the brain stem?
3. How does the nerve reach the dorsal position where it decussates?

Frazer (1928-1929) considered the problem from an embryological standpoint, and believed that the course of the nerve was affected by developmental occurrences in the region of the isthmus rhombencephali. He described and figured a displacement forwards

or invagination of the basal lamina of the hind-brain towards the mid-brain where it is folded down against the floor (basal) lamina of the latter producing an intra (basal)-laminar sulcus. The region where this occurs is the isthmus rhombencephali. Furthermore Frazer believed that the alar lamina extended on the outer side of the basal lamina of the isthmus, thus enabling the basal lamina which is displaced forwards, to form the lateral wall of the isthmus. In the 12 mm. embryo he found that where the basal lamina of the hind-brain continued rostrally to form the lateral wall of the isthmus, it produced a distinct rounded border in which was "situated the trochlear nucleus from which the nerve fibres run up (*i.e.*, dorsally) through the basal lamina to decussate." In the light of this rostral projection of the basal lamina, Frazer explained the deep course of the trochlear nerve, and stated that the nerve originally emerged from the basal lamina of the ventral aspect of the hind-brain. As the basal lamina is carried forwards to form the lateral wall of the isthmus, it takes the nerve with it. In this way the nerve gains the dorsal aspect of the isthmus where it emerges. He says "the issuing nerve is essentially ventral, although actually dorsal when the isthmus is thus completed."

Frazer attempted to explain the decussation of the trochlear nerves by likening them to the efferent spinal nerves which are composed mostly of homolateral fibres, but contain a few contralateral fibres, the latter of which decussate. He suggested that the fourth nerve consists entirely of contralateral fibres which decussate in the orthodox fashion.

Kappers, Huber and Crosby (1936) believe that the course of the trochlear nerve is due in part to a shifting forwards of the cells of origin during development. Hamilton, Boyd and Mossman (1945) state that the "atypical course of the fourth nerve, which apparently is to be regarded as somatic efferent in nature, has never been adequately explained."

From a study of a series of human embryos it became apparent that Frazer's explanation of the dorsal course of the trochlear nerves might profitably be reinvestigated. Regarding his suggested reasons for the decussation, it is relevant to refer to Gaskell's hypothesis. Reviewing the work of Beck, of Fürbringer and of Hoffman, Gaskell (1908) regarded the vertebrate eye muscles as comparable with the dorso-ventral prosomatic segmented muscles of the invertebrates. In the scorpion each of the pair of muscles known as the anterior dorso-plastron muscles crosses the mid-dorsal line to reach its insertion and carries its nerve supply with it. Gaskell suggested that these muscles become the superior oblique muscles of the vertebrates, and that herein lies the explanation of the trochlear route.

Present investigations

The trochlear complex as seen in a series of human embryos and foetuses (4 mm. onwards) is described in the following pages. An account of the development of the isthmus rhombencephali is also included. The question how does the trochlear nerve reach the dorsal region of the brain stem is answered and the nature of the decussation is described. The investigation has not revealed why the decussation occurs or why it takes place on the dorsal aspect of the brain stem.

Since the publication of the paper referred to on p. 257, the author has obtained further early embryos in excellent condition. All embryos used in these investigations have been graded according to their length, and, where possible in accordance with the history. In some cases, however, the age estimate may not correspond strictly to the stage of development. For example in a 9 mm. and a 10 mm. embryo, recently received, both the oculomotor and trochlear nerves were found to have emerged from the brain stem. In a previous 10 mm. embryo neither was observed to have reached its superficial origin.

The earliest embryo available measures 4 mm. (estimated age between three and four weeks). Fore-, mid- and hind-brain are differentiated, and the optic vesicles form lateral projections from the fore-brain vesicle. A slight constriction exists between the rhombencephalon and mesencephalon which denotes the position of the future isthmus (Fig. 1). The otic vesicles appear in section as round cavities lying lateral to the hind-brain and separated from the surface ectoderm. On the cephalic aspect of each otocyst is the primitive acoustic ganglion composed of round or spindle-shaped cells with short processes which do not appear to extend either peripherally or centrally.

At a more rostral level is a larger ganglionic mass placed just caudal and lateral to the primary head vein (anterior cardinal vein). This is the trigeminal ganglion and, like the acoustic ganglion, it shows no connection with the neural tube. These ganglia indicate respectively the myelencephalic and metencephalic portions of the rhombencephalon. The thin roof plate of the rhombencephalon is present. The ganglia of the ninth and tenth cranial nerves and the posterior root ganglia of the spinal nerves are well formed, but none appears to be joined to the neural tube. It must be mentioned, however, that in such delicate tissue the continuity between the ganglia and the neural tube may have been destroyed as a result of death and histological preparation.

The neural tube consists throughout of ependymal, mantle and marginal layers. The mantle layer is similar in all parts, and

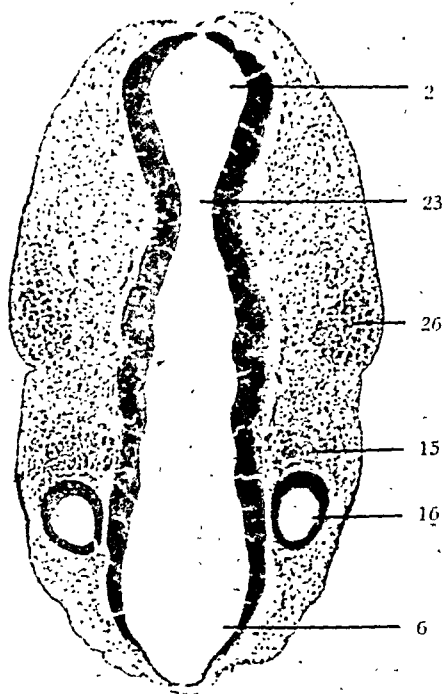


FIG. 1.

Section through head end of a 4 mm. embryo. $\times 50$.

possesses seven to ten layers of undifferentiated (indifferent) cells. The roof (alar) and floor (basal) plates exhibit only the ependymal layer with one or two rows of cells. In no part of the neural tube is there any concentration of cells in the basal lamina indicative of efferent nuclear formation, and no efferent nerves, either spinal or cranial are recognisable.

An embryo of 9 mm. length (about $4\frac{1}{2}$ weeks) sectioned in the plane of the long axis of the rhombencephalon, gives an interesting picture of the developing brain stem. It will be seen from Fig. 2a that, owing to the neural flexures, the section illustrated in Fig. 2b shows the alar region of the metencephalon bounding the fourth ventricle in its cephalic half. Already a thickening of this alar plate has occurred on each side, forming the cerebellar rudiments. The thickening affects the two lateral portions of the alar plate, and each forms an intraventricular projection. As the fourth ventricle narrows at its cephalic extremity, the two (thickened)

cerebellar plates become approximated but do not meet, a small unthickened median portion of the (cerebellar) alar plates persisting at the apex of the fourth ventricle. This thin median plate becomes the superior medullary velum and Fig. 2*c* shows its continuity at the rostral apex of the fourth ventricle with the alar plate of the mesencephalon, a tangential section only of the latter being

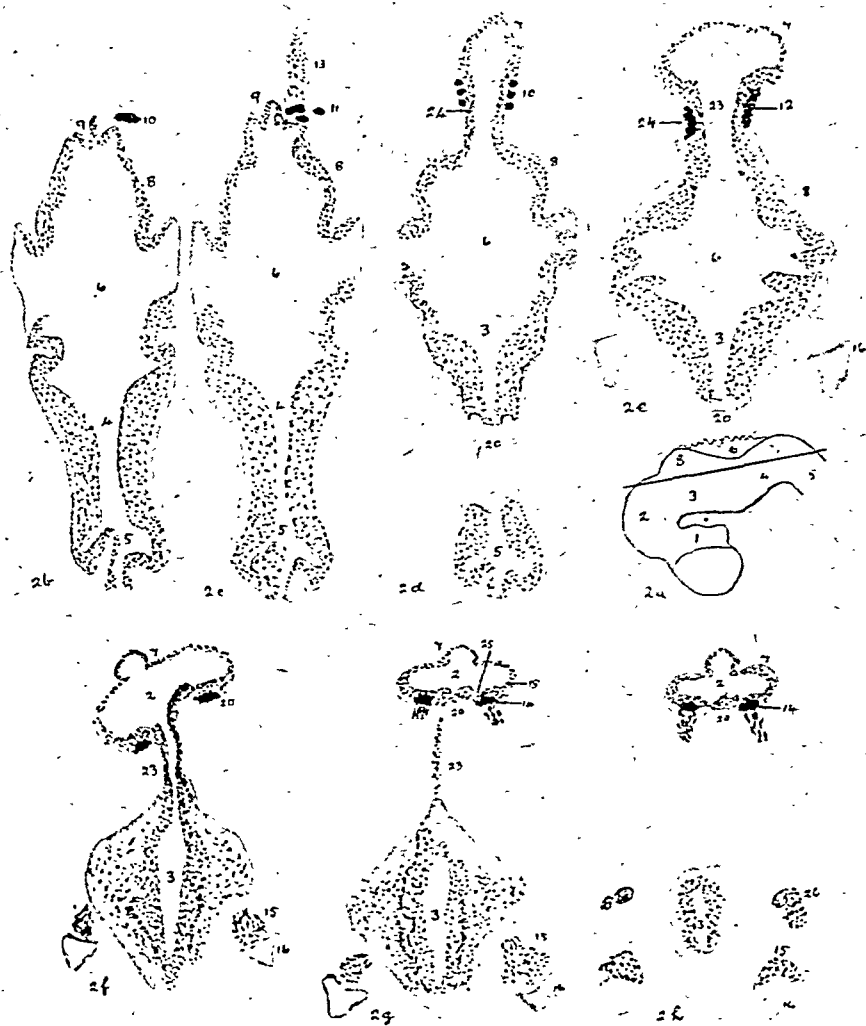


FIG. 2.

Serial sections through the brain stem of a 9 mm. embryo.

included. It is at the junction of the median portions of the metencephalic and mesencephalic alar plates that the decussation of the trochlear nerves is seen (Fig. 2c).

In the sections still deeper into the rhombencephalon, the opening up of the apical recess of the fourth ventricle into the wider cavity of the mesencephalon is visible (Fig. 2d). The narrow communication between the metencephalon and mesencephalon is the isthmus. Another point of interest is apparent in Fig. 2d. Here the lateral alar thickenings of the metencephalon (which form the cerebellar plates) continue rostrally side by side, and in this way become continuous with the lateral walls of the narrow isthmus. In each lateral wall the trochlear nerve can be identified lying on the outskirts of the mantle layer. Subsequent sections at more ventral levels show that the lateral walls of the isthmus finally merge into the basal plate of the mesencephalon where the narrow isthmus channel gives place to the wider mesencephalic cavity (Figs. 2e, f, g and h).

Thus the rostral extremities of the lateral walls of the isthmus join the floor or basal plate of the mesencephalon, forming the lateral parts. The intra (basal)-laminar sulci, described by Frazer, demarcate the superadded lateral parts of the floor plate from the median part which corresponds to the true basal lamina of the mid-brain.

Thus in this 9 mm. embryo there can be seen :—

1. Metencephalic alar thickenings on each side of the cephalic part of the fourth ventricle forming the cerebellar plates.
2. Mid-line apical alar region not thickened, but joining the rostral extremities of the cerebellar plates together and forming the superior medullary velum. Here the trochlear decussation occurs.
3. Traced rostrally, the metencephalic alar thickenings continue as the lateral walls of the isthmus.
4. Still more rostrally, the lateral walls of the isthmus merge into the floor plate of the mesencephalon.

In this series it is apparent that in the lateral walls of the isthmus there is continuity between tissue derived from metencephalic alar and mesencephalic basal plates. This junctional tissue forms the lateral isthmus walls, while the continuity of the superior medullary velum and the mesencephalic alar lamina closes the isthmus dorsally. Owing to the plane of the sectioning, the floor of the isthmus is not seen.

The nucleus of the trochlear nerve is recognisable as a small group of slightly larger differentiating cells lying on the outskirts

of the mantle layer in the lateral wall of the isthmus (Fig. 2e). The lateral isthmic walls blend with the mesencephalic basal plate and form the lateral parts of its floor, and here the oculomotor nuclei are situated. Thus the oculomotor and trochlear nuclei are in series, but at this 9 mm. stage, the former is located in the mesencephalon and the latter in the isthmus. From the trochlear nucleus a tiny nerve bundle runs along the edge of the mantle layer and can be followed in the lateral wall of the isthmus in a caudal direction to the apex of the fourth ventricle where the decussation occurs in the superior medullary velum and the nerve gains its superficial origin (Figs. 2e, d and c).

Sectioning of an embryo of 13 mm. length in the coronal plane from the vertebral column forwards, yielded a dorso-ventral or alar-basal series of the brain stem (Fig. 3a). It will be seen that the basal plate of the metencephalon forms the floor of the fourth ventricle, and the alar region, thickened by the intra-ventricular cerebellar plates, bounds the ventricle laterally (Fig. 3b). The cerebellar rudiments exhibit differentiation, and ependymal, mantle and marginal layers are wide and distinct. The thick cerebellar plates incline towards each other and towards the mid-line, but do not meet. At the cephalic apex of the fourth ventricle they are linked by alar tissue not included in the cerebellar thickening. This thin tissue is the superior medullary velum (Fig. 3b). Sections at a more cephalic level contain the decussation of the trochlear nerves in the velum and their emergence on the dorsal surface of this region (Fig. 3c). The more rostral part of the decussation occurs where the superior medullary velum is meeting the alar plate (tectum) of the mesencephalon (Figs. 3c and 4).

Traced in the cephalic direction, the two lateral alar (cerebellar) plates of the metencephalon, which are separated from each other at the apex of the fourth ventricle by the superior medullary velum, remain distinct, but lie side by side, leaving a slit-like cavity between them as the mid-brain is approached. The region with the narrow cavity is the isthmus, and the rostral continuation of each metencephalic alar (cerebellar) plate comes to form a lateral wall of the isthmus (Fig. 3d).

The narrowing of the isthmic cavity is the result of the close approximation of the lateral walls. In consequence, the basal lamina of the isthmus is compressed so that part of it actually forms the most ventral part of the lateral isthmic wall. But the basal lamina of the isthmus is continuous rostrally and caudally with that of the mesencephalon and metencephalon respectively, although in both these regions it is expanded by the widening of the cavities. The elongation of the isthmic cavity is exaggerated owing to obliquity of the sections.

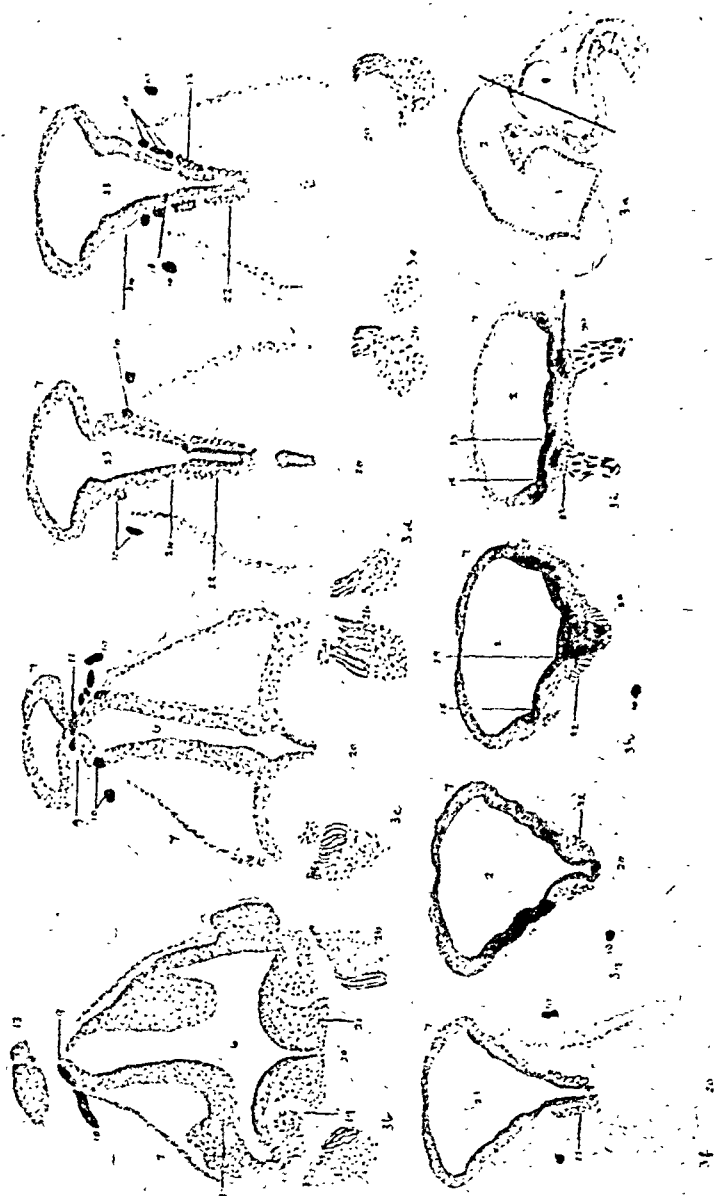


FIG. 3.

Serial sections through the brain stem of a 13 mm. embryo.

At a more cephalic level the cavity of the isthmus joins the wider cavity of the mesencephalon, and the lateral walls of the isthmus merge into the basal plate of the mid-brain forming its outer parts. The most ventral part of the lateral wall of the isthmus is really basal lamina (Fig. 3e), and here is situated the trochlear nucleus in series with the oculomotor nucleus (Figs. 3e and 5). Thus the trochlear nucleus lies in the lateral isthmic wall, but this

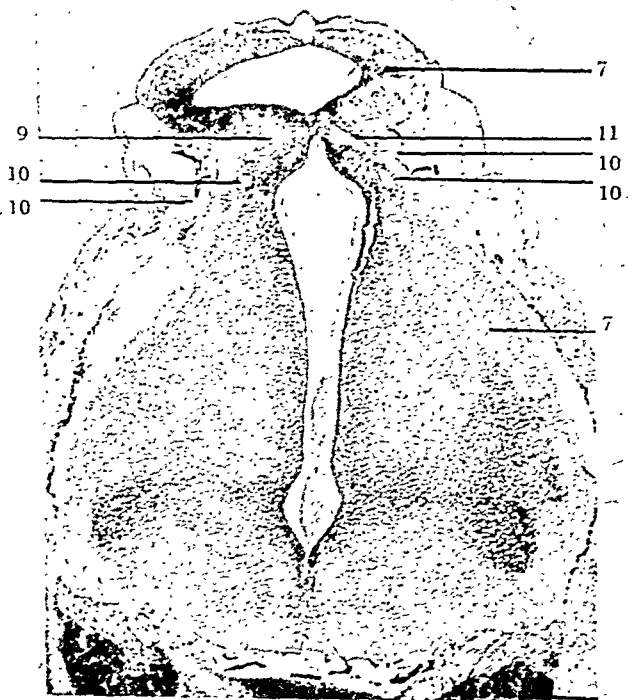


FIG. 4.

Region of isthmus of a 13 mm. embryo. (See Fig. 3c). $\times 30$.

is only because the basal lamina has been pushed into this position by the elongation of the lateral isthmic walls and consequent narrowing of the contained cavity.

From the trochlear nucleus a bundle of fibres runs dorsally in the lateral walls of the isthmus and is then carried caudally to the apex of the fourth ventricle where it decussates with its fellow in the superior medullary velum and emerges superficially (Figs. 3e, d, c and b).

From this series of a 13 mm. embryo it would appear that there is a continuity of tissue between the cerebellar (alar) plates of the

metencephalon, the lateral wall of the isthmus, and the floor (basal) plate of the mesencephalon. This continuity ultimately enables the cerebello-mesencephalic fibres (superior cerebellar peduncles) to follow the route they do, and in the embryo of 19 mm. these fibres have traversed this pathway into the basal plate of the mid-brain where they decussate (Cooper, 1946 (b)). This then

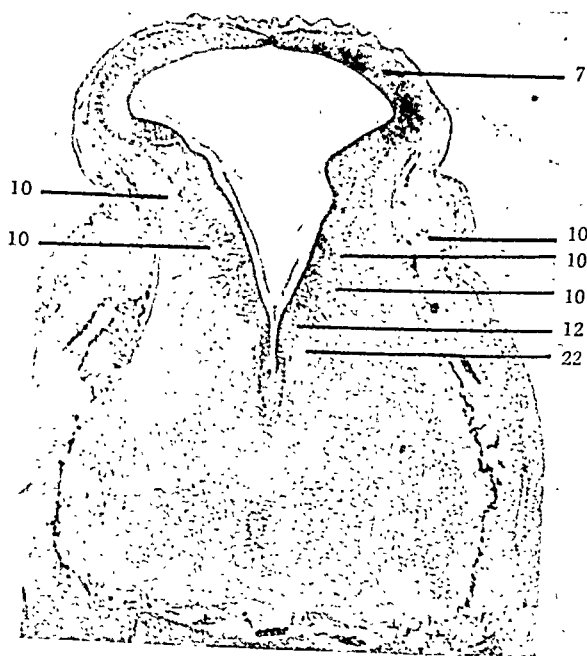


FIG. 5.

Cephalic end of isthmus of a 13 mm. embryo. (See Fig. 3c). $\times 30$.

is the essential difference between Frazer's account of the development of the isthmus and the present findings. He described the lateral wall of the isthmus as being formed by a thrust forwards of the basal lamina of the metencephalon towards the mesencephalon. The present investigation seems to show that the lateral wall of the isthmus unites the rostral continuation of the metencephalic alar (cerebellar) plate to the mesencephalic basal plate, and thus provides a route for the cerebello-mesencephalic fibres.

An embryo of 15 mm. sectioned from the highest part of the mesencephalon gives another view of the isthmus and the trochlear nerves (Fig. 6a). In the sections through the caudal extremity of the metencephalon, the alar and basal plates are lying in the

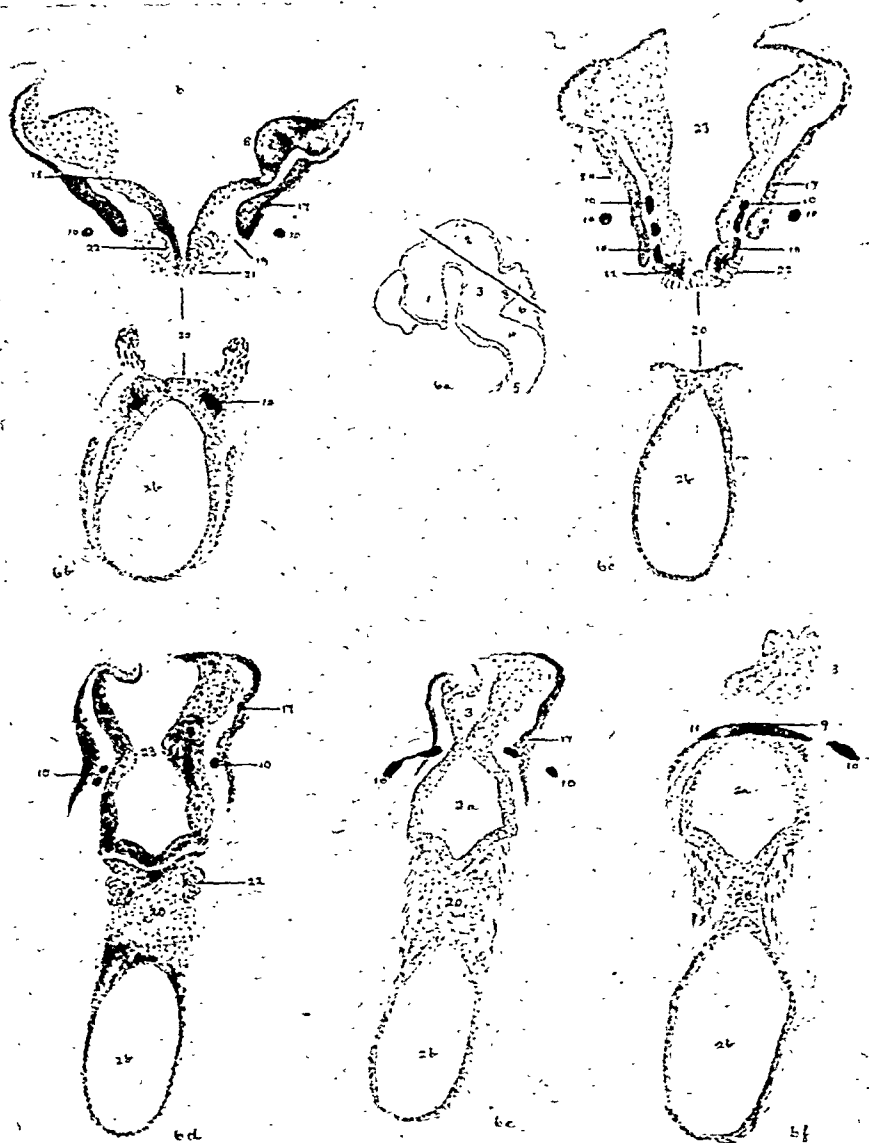


FIG. 6.

Serial sections through the brain stem of a 15 mm. embryo.

floor of the fourth ventricle, the roof being ependymal (Fig. 6b). The proliferation of neuroblasts is greater in the alar than in the basal plates, and in the former a dorsally directed intra-ventricular enlargement forms the cerebellar rudiment. In addition a spread of cells from the alar portion has progressed ventro-medially towards the basal plate. The basal portion has given rise to a cell

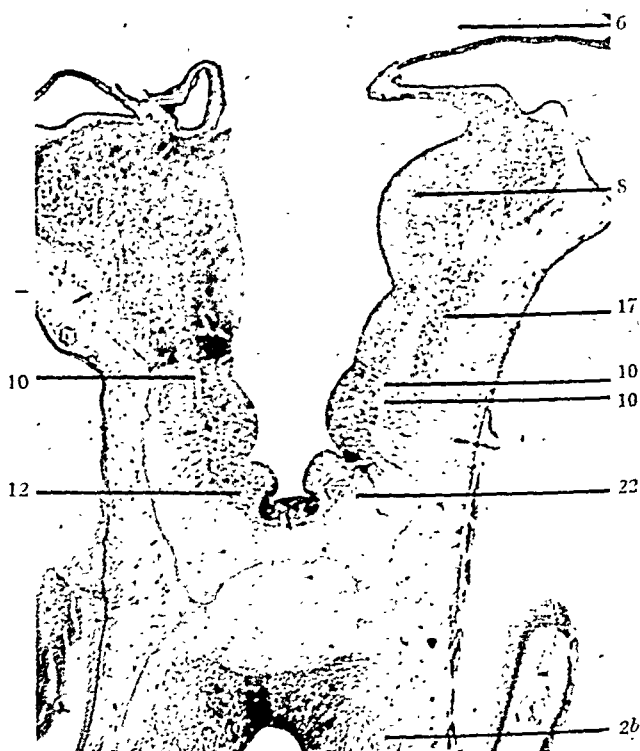


FIG. 7.

Region of isthmus of a 15 mm. embryo. (See Fig. 6c). $\times 30$.

spread which is limited to the median part of the floor of the fourth ventricle. These alar and basal spreads appear to be separated by a relatively cell-free zone sufficiently well defined to justify the assumption that it is an inter-laminar zone (Fig. 6b). It does not correspond, however, to the indentation on the ependymal aspect which is usually called the interlaminar sulcus (sulcus terminalis). It is suggested, therefore, that the junction of the alar and basal plates corresponds to the cell-free zone and not to the interlaminar sulcus. If this be true, then the alar plate is thickened

in its larger lateral part to form the cerebellar rudiment, while its smaller medial part adjacent to the basal plate is not enlarged. In other words the interlaminar sulcus is a depression resulting from the cerebellar enlargement of the lateral part of the alar plate.

Following the sections serially in a dorsal direction, it is found that the alar plates gradually approach each other until they lie parallel and bound a narrow cavity (Figs. 6c and 7). This region corresponds to the isthmus rhombencephali. The alar plates come to form the lateral walls of the isthmus, while the basal plate is pressed down to bound the ventral recess of the isthmic cavity. Furthermore, since the recess is exceedingly narrow, part of the basal plate assumes a lateral position and comes to lie in the most ventral part of the lateral isthmic wall, the rest of this wall being formed by alar plate (Figs. 6c and 7).

It is in this position that the trochlear nucleus is found, lying in the basal lamina of the isthmus, but where this lamina is forming the most ventral part of the lateral isthmic wall (Figs. 6c and 7). Traced more dorsally (Figs. 6d and e, and 8) the two lateral isthmic walls finally meet, thus separating off an overhanging portion of the metencephalic cavity from the isthmic cavity which now begins to widen out to join the mesencephalic cavity. During this widening out process, the isthmic basal lamina is spread out again, and once more lies in the floor plate when the mesencephalon proper is reached (Fig. 6f).

It is apparent from Figs. 6e and f, and 8 that the meeting of the lateral isthmic walls coincides with the superior medullary velum which continues into the alar (roof) plate of the mesencephalon. Also it will be seen that the lateral walls of the isthmus are continuous with alar tissue which forms the cerebellar plates more caudally. Furthermore owing to the mesencephalic flexure, the mesencephalic basal lamina comes into line with the lateral wall of the isthmus and this continues into the lateral part of the basal lamina of the mid-brain (Fig. 6a).

The trochlear nucleus in this series appears as a semilunar mass of neuroblasts lying in the compressed basal plate at the ventral extremity of the lateral isthmic wall. The neuroblasts are distinctly larger than any other cells (Figs. 6c and 7). From the cells in the nucleus, fibres turn dorsally and immediately enter the lateral isthmic wall proper which is mainly alar in origin (Figs. 6c and 7). Thence the nerve passes in the lateral wall of the isthmus to that position where the two lateral walls meet at the superior medullary velum. Here the two nerves decussate and emerge at their superficial origin (Figs. 6e and f, and 8).

Thus, judging from serial sections of early embryos cut :—

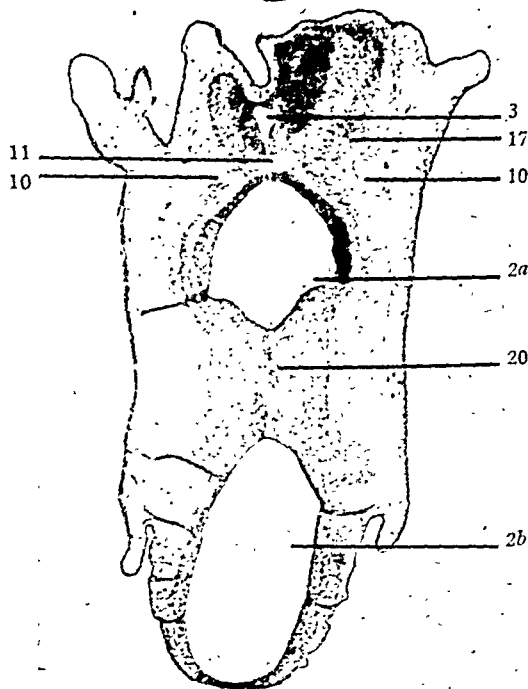


FIG. 8.

Cephalic end of isthmus of a 15 mm. embryo. (See Fig. 6c). $\times 20$.

1. in the plane of the long axis of the rhombencephalon;
2. in the dorso-ventral (alar-basal) plane; and
3. from the highest part of the mesencephalon, the following points are demonstrable:—

1. The metencephalic alar tissue is continued rostrally in the lateral isthmic walls to the mesencephalic basal plate. (By the 19 mm. stage this route is traversed by cerebello-mesencephalic fibres (superior cerebellar peduncles)).

2. The trochlear nucleus lies in the lateral isthmic wall, but in the most ventral part which is formed by the basal plate. This plate is squeezed into this position by the alar tissue which forms the bulk of the lateral wall of the isthmus.

3. The trochlear fibres pass dorsally and caudally within the lateral wall of the isthmus to the alar plate of the metencephalon, these two structures being continuous. Thus the trochlear nerve

reaches the dorsum of the isthmus and decussates in the superior medullary velum and escapes on the dorsal aspect of the brain stem.

4. The position where the lateral isthmic walls join the mesencephalic basal lamina is marked by the intra (basal)-laminar sulcus.

5. In the metencephalon the line of demarcation between the alar and basal plates apparently does not correspond to the inter-laminar sulcus, but it is indicated by a cell free zone.

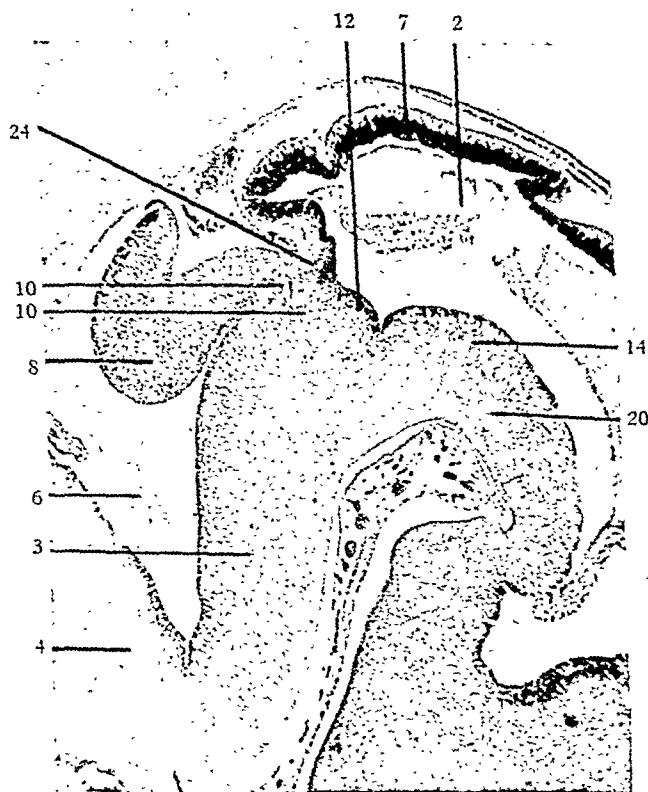


FIG. 9.

Parasagittal section through the brain stem of a 22 mm. embryo. $\times 25$.

Figure 9 is a parasagittal section through the brain of an embryo of 22 mm. It has passed through the lateral wall of the isthmus. The fibres of the superior cerebellar peduncles sweep forwards and ventrally through the lateral wall of the isthmus to the basal plate of the mid-brain where they decussate and join the red nucleus. The oculomotor nucleus is recognisable in the tegmental (basal) region of the mid-brain. Just caudal to it is the trochlear nucleus lying in the basal plate and overhung by the enlarging

tectum of the mid-brain. Fibres of the trochlear nerve are seen to be running from the nucleus dorsally and caudally, and by following the direction of the cerebello-mesencephalic fibres, reach their decussation in the superior medullary velum.

In this series the nucleus of the trochlear nerve is apparently located in the isthmus in embryos up to 22 mm. length, but one point must be borne in mind. An accurate estimation of the extent of the isthmus is difficult owing to the modification produced by the neural flexures as age increases, and the resulting variation in the planes of serial sections.

The above observations provide a possible answer to the question how does the trochlear nerve reach its dorsal decussation? It has been shown that the trochlear nucleus at first lies in the isthmus, and the nerve issuing therefrom gains the dorsal region of the metencephalon in tissue which extends between the mesencephalic basal and metencephalic alar plates. It would seem that this is a route offering little resistance to the course of the nerve, and it also becomes the pathway for the fibres of the superior cerebellar peduncles. If the nerve pursued a ventral course it would ultimately need to penetrate through the fibres of the superior cerebellar peduncles which soon form a dense mass in the basal plate of the mesencephalon.

The next point to be considered is the final orientation of the trochlear nucleus in the caudal region of the mesencephalon. From Fig. 9 it will be seen that already the tectal (alar) plate of the mesencephalon has grown to a greater extent than the basal, and that it is overhanging the fore part of the isthmus. It is evident that when the extra-ventricular growth of the cerebellum occurs, it encroaches on the isthmus from behind. Examination of the adult brain stem justifies the statement that growth of the isthmus lags behind that of the mesencephalon and metencephalon, and its identity is somewhat obscured. It may be that the enlarging mesencephalon tends to draw the isthmus in the rostral direction, while the metencephalic development pushes it forwards. In addition the sweep of the fibres of the superior cerebellar peduncles from the metencephalon to the mesencephalon which commences at the 19 mm. stage, may carry the trochlear nucleus to a more cephalic position. As development proceeds, decrease in the degree of the mesencephalic and pontine flexures, which affect the basal plates, will help to establish the trochlear nucleus in a more cephalic position relative to the enlarging mesencephalic tectum.

The last point to be mentioned is the nature of the trochlear decussation. In all embryonic, foetal and adult specimens examined, the trochlear nerve seems to form a compact bundle as the fibres emerge from the nucleus. This bundle pursues its dorso-caudal

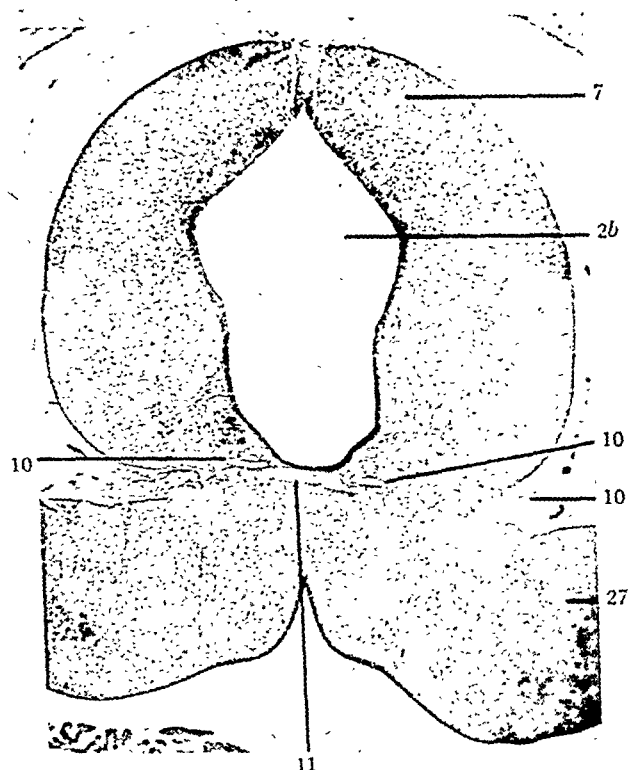


FIG. 10.

Section through the trochlear decussation of a 37 mm. embryo. $\times 23$.

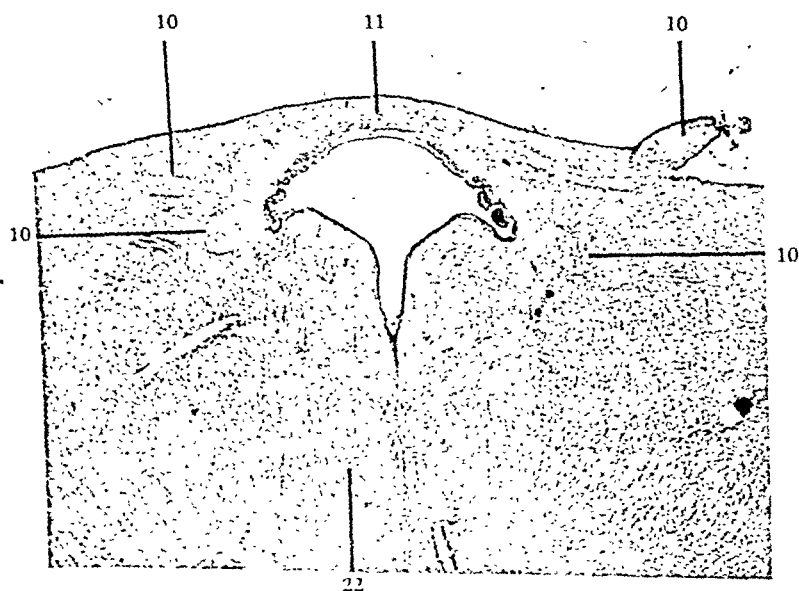


FIG. 11.

Section through the trochlear decussation of an adult. $\times 10$.

course and a simple crossing of the two bundles occurs. There does not appear to be the thorough intermingling of fibres as in the great fibre decussations of the superior cerebellar peduncles, pyramids or medial lemnisci. Invariably a single bundle emerges at the surface (Figs. 4, 8, 10 and 11).

Conclusions

1. The two metencephalic alar plates (cerebellar rudiments) approximate closely to each other and to the mid-line at the rostral apex of the fourth ventricle. Here they are linked together by a thin stratum of tissue which becomes the superior medullary velum.
2. These metencephalic alar plates are continued rostrally around the lateral aspect of the neural tube at the position of the isthmus rhombencephali, and thus form its lateral walls.
3. Traced in the cephalic direction, the lateral isthmus walls join the basal plate of the mesencephalon on either side when the narrow cavity of the isthmus expands into the mid-brain cavity.
4. Thus a continuity of tissue exists between the metencephalic alar (cerebellar) plate, the lateral wall of the isthmus and the mesencephalic basal or floor plate. The fibres of the superior cerebellar peduncles follow this route from the cerebellum to the red nucleus at the 19 mm. stage of the embryo.
5. The trochlear nucleus originates in series with the oculomotor nucleus but in the basal plate of the isthmus. As the lateral walls of the isthmus link the metencephalic alar to the mesencephalic basal plates, the basal plate of the isthmus becomes compressed until it assumes a position at the ventral extremity of the lateral wall of the isthmus, and it is here that the trochlear nucleus is situated at first. The fibres from the nucleus form a bundle which turns dorsally at once and thus gains an alar situation in the lateral wall of the isthmus. The nerve then proceeds through the lateral wall of the isthmus caudally to the superior medullary velum where the decussation occurs.
6. Beyond 22 mm. (7 weeks) the trochlear nucleus is orientated in the mid-brain and suggestions are put forward to explain this apparent shifting forwards.

The author wishes to express thanks to Professor G. A. G. Mitchell of the Department of Anatomy in the University of Manchester for his generous advice and criticism; to Professor F. Wood Jones, F.R.S., for his interest and help in the comparative

aspect of the problem; to Professor D. Dougal, Dr. G. Stewart Smith and Dr. Mary Evans for the embryos and foetuses; and to Mr. H. Gooding for preparing the photographs.

KEY TO ILLUSTRATIONS.

- | | |
|---|---|
| 1. Diencephalon. | 13. Tangential section of mesencephalic alar plate. |
| 2. Mesencephalon (a) caudal level;
(b) cephalic level. | 14. Oculomotor nucleus and/or nerve. |
| 3. Metencephalon. | 15. Acoustic ganglion. |
| 4. Myelencephalon. | 16. Otocyst (otic vesicle). |
| 5. Spinal cord. | 17. Alar spread. |
| 6. Fourth ventricle and/or its taenial roof. | 18. Interlaminar sulcus. |
| 7. Alar plate. | 19. Interlaminar cell-free zone. |
| 8. Cerebellar rudiment. | 20. Basal plate. |
| 9. Superior medullary velum. | 21. Basal spread. |
| 10. Trochlear nerve. | 22. Medial longitudinal bundle. |
| 11. Decussation of trochlear nerves. | 23. Isthmus rhombencephali. |
| 12. Trochlear nucleus. | 24. Lateral wall of isthmus. |
| | 25. Intra (basal)-laminar sulcus. |
| 26. Trigeminal ganglion and nerve. | |
| 27. Superior cerebellar peduncle. | |

The use of a hand lens is recommended in examining the photographs.

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COLOUR VISION IN THE CONSULTING ROOM

BY

FRANK R. NEUBERT

GUERNSEY

IN 1941 an analysis of the colour-vision reports on 2,484 subjects suggested that the difference in the findings on colour-plates and colour-lanterns would form an interesting subject for investigation.

Such an investigation began, and during the following four years 43,395 subjects were examined: 40,380 males and 3,015 females.

Only two defectives (both unsafe) were found among the latter, and the final figures were restricted to the male sex only.

Many subjects were seen on whose colour sense opinions had differed, and an attempt was made to determine (a) why such defects could not be assessed easily and positively (b) why results on colour plates and colour lanterns did not always agree, and (c) whether an improvement could be made in clinical examination apart from the use of laboratory methods.

Colour Perception with Aviation Model

The terms "normal," "unsafe," "safe," "green-blind," and "red-blind," although scientifically inaccurate, are understood by all, and are used in their commonly accepted meaning in this résumé of the work done.

All the 40,380 men were asked to read the original Ishihara Colour Plates, 8th Edition, and each one who made a mistake suggesting a defective colour sense—2,235 in all—was investigated on a lantern (colour perception unit—aviation model) and by other experimental methods.

A group of 1,654 subjects was first examined on plates and lantern and the results compared.

TABLE I.
Examination of 1,654 previously untested subjects on Plates and Lantern.

					Ishihara Plates	Lantern Unit	
Normal	1,585	1,623	
Safe	20 (1.21%)	3	(0.015%)
Unsafe	Red-Blind	8	49 (3.1%)	3	28 (1.75%)
	Green-Blind	38		22	
	Red and Green-Blind	3		3	
Total	1,654	1,654	

The difference between these findings was too great to be ignored, and a second group of 431 subjects, who were classified as unsafe by the plates, were examined on the lantern: 135 (31.3 per cent.) passed. From this it appeared that the plates were approximately one third more sensitive than the lantern in detecting defects in the colour sense.

TABLE II
Comparison of Defective Subjects by Ishihara Test and Lantern

Ishihara	Lantern	
	Colour Defective Unsafe	Colour Defective Safe
Red-Blind ... 178	131	47
Green-Blind ... 236	153	83
Red & Green-Blind 17	12	5
Total ... 431	296 (68.7%)	135 (31.3%)

The manner in which the colour plates were read made it seem advisable to record the individual mistakes, and 1,573 unselected men were asked to read, without hesitation, a group of figures in an American series of plates (mentioned later).

686 (43.7 per cent.) of them were certainly normal or definitely defective. 532 (33.8 per cent.) of them made mistakes which were corrected at a second attempt. 355 (22.5 per cent.) of them made mistakes which were not corrected after two or more attempts, although such errors did not come into the category "Colour defective" according to the explanation book.

For example, if plates Nos. 1 and 2 (figures 89 and 43) were read as 89 and 45, altered to 89 and 48, then corrected to 89 and 43, the mistakes would appear due to observation rather than a defect in the colour sense and such cases were not classified as defective. This is in agreement with the instructions.

Pseudo-iso Plates

The original Ishihara plates began to fade, and tests were commenced with the series issued by the American Optical Company: The pseudo-isochromatic plates for testing colour perception.

Each series contains a pair of plates showing the numerals 45 and 73 which are intended to be similar: Ishihara Nos. 20 and 21, A.O.C. Nos. 33 and 34. These are supposed to be read only by the majority of the red-green blind, "but the majority of the normal and the totally colour-blind can hardly see them" (Ishihara 1939). The results of 11,348 readings were analysed.

By Ishihara standards 445 (3.9 per cent.) were definitely colour-blind, 370 (3.2 per cent. Group A) read the Japanese figures, but only 193 (1.7 per cent. Group B) read the American ones, a difference of 47 per cent.

TABLE III

Comparison of 445 defective subjects by Ishihara and American Plates.

	Ishihara Plates	American Plates
Read (unsafe)	370 (83.0%) Group A	193 (43.4%) Group B
Not read (safe)	75 (17.0%)	252 (56.6%)
Total ...	445	445

The 370 subjects (Group A) were examined on the lantern and 124 (33.5 per cent.) of them were found to be safe.

The 193 subjects (Group B) were examined similarly and 55 (29.5 per cent.) were found to be safe.

Examination of these results showed that, whereas only two subjects could read the American and not the Japanese figures, 200 were able to read the Japanese but not the American. The lantern results of these cases are compared in Table IV.

TABLE IV

Classification by Lantern of subjects in Table III who read Ishihara plates Nos. 20 and 21 and American Plates Nos. 33 and 34.

Lantern Classification	Ishihara Unsafe	American Unsafe
Safe ...	124 (33.5%)	55 (29.5%)
Unsafe ...	246 (66.5%)	138 (70.5%)
Total ...	370 (Group A)	193 (Group B)

This not inconsiderable difference emphasises the exceptional difficulty in producing dyes which are suitable for colour-vision testing.

When a British reprint of the Ishihara plates was issued, a comparison was made between it and the Japanese set, but as so many normal subjects read without much difficulty the figures which only the colour-blind were intended to see, the set was not included in the investigation and only the original series was used.

The Ishihara test claims to separate by means of plates Nos. 22, 23, 24, and 25, the "red-blind" from the "green-blind."

A detailed examination on the lantern was made of those defective classified by this means.

TABLE V

Ishihara	Lantern Test			Passed Lantern Test
	Failed to recognise Red only	Green only	Red and Green	
Red-Blind ... 116	7	53	35	21
Green-Blind ... 194	16	72	35	71
Red and Green-Blind 27	3	11	6	7
Total ... 337	26	136	76	99

Out of the Ishihara "red-blind" cases, only 7 failed to recognise only red on the lantern, whereas 35 were unable to recognise both red and green, and 21 passed the test.

Out of 194 who were Ishihara "green-blind," 72 were unable to recognise only green on the lantern, whereas 35 failed to recognise both red and green, and 71 passed the lantern test.

It has been claimed that any subject classified by these figures will eventually fail with the lantern if examined persistently, but this was not found to be the case.

TABLE VI

An analysis into "Safe" and "Unsafe" on the lantern of 368 subjects classified according to Ishihara plates as Colour Defective Unsafe.

	Red-Blind	Green-Blind	Red and Green-Blind	Total
Ishihara Plates	122	223	23	368
Lantern—Safe...	11	81	6	98
" Unsafe	111	142	17	270
				368

A total of 368 cases was classified, of whom 122 were found to be red-blind and 223 were found to be green-blind. 92 of these cases passed the lantern test after having been examined persistently.

During these tests a comparison was made between the aviation model colour perception unit and the Edridge-Green colour lantern; 190 defective subjects were examined on each, using the technique outlined in the respective instructions.

The comparison suggests an inherent weakness in this type of lantern.

TABLE VII

The percentage comparison between the Aviation Model and Edridge-Green Lantern.

<i>Unsafe</i> Aviation Model	<i>Unsafe</i> Edridge-Green	47.4%
<i>Unsafe</i> Aviation Model	<i>Safe</i> Edridge-Green	15.8%
<i>Safe</i> Aviation Model	<i>Unsafe</i> Edridge-Green	15.8%
<i>Safe</i> Aviation Model	<i>Safe</i> Edridge-Green	21.0%

PART II

This examination and comparison of the usual clinical methods of determining the state of the colour sense suggested that more attention should be devoted to lantern tests and a considerable number of experimental lanterns were made. The original postulates of Guttman, translated in Parsons' "Colour Vision," suggested the lines along which these experiments should develop.

Guttman found that those with defective colour perception differed in seven respects from the normal.

(1) They required a smaller difference in the yellow but a greater difference in the green for discrimination.

(2) They were more dependent upon luminosity for hue discrimination.

(3) They developed a capacity for distinguishing differences in luminosity which they translated into hue.

(4) They required a larger area of retinal stimulation.

(5) They required a longer time to make their decision.

(6) Their colour sense was more readily fatigued (successive contrast).

(7) They had an increased sense of simultaneous contrast.

The chromatic threshold

Parsons states that the appreciation of colours only occurs with lights of moderate or high intensity (photopic vision). If a spectrum of low intensity is seen with the dark adapted eye it appears as a grey band, differing in brightness in different parts (scotopic vision).

It would appear from this that, with the very low threshold for red, an exaggeration between the appreciation of red and green, or between red and yellow, would be made by a defective person if two or three colours were presented simultaneously to the photopic eye by means of a lamp with controlled illumination. A rheostat was fitted to all lanterns.

Size of lantern aperture

According to Guttman's fourth postulate, a colour defective person requires a considerably larger area of stimulation for the perception of hue.

The duplicity theory of vision asserts that the retinal cones are responsible for the perception of colour. The cones are most numerous at the fovea centralis, where rods are absent, and decrease proportionately peripheralward.

The measurements of the so-called "rod-free area" are:

Fovea Centralis	...	0.24 to 0.3 mm. in diameter	(55' to 70')
Rod Free area	...	0.80 mm. (3°3')
Macula	...	1.0 to 3.0 mm. (4° to 12°)

In order to fall within these areas, an object at a distance of 6 metres would have to be:—

Fovea Centralis	...	9.6 to 12 mm. in diameter.
Rod free area	...	32 mm. /
Macula	...	40 to 120 mm.

In order to stimulate an individual cone 0.004 mm. diameter, an object must be 1.7 mms. at 6 metres, which is the exact size of each element of a Snellen's 6/6 test-type letter.

An examination was made at 6 metres with the 1 millimetre aperture of the lantern of 313 colour defectives who had been classified on the 5 mm. aperture.

139 had been passed as "safe." 29 (20.9 per cent.) of these failed to recognise the red or green, or both the red and green 1 millimetre aperture.

174 had been considered as "unsafe." 77 (36.8 per cent.) of these were able to recognise both the 1 millimetre red and green.

TABLE VIII

Comparison of the effect of Aperture Sizes.

LANTERN			COLOUR
Classification of Cases using the 5 mm. aperture	Recognised 1 mm. aperture	Failed on 1 mm. aperture	Analysis of Failures
C.D. Safe ... 139	97 (79.1%)	29 (20.9%)	Red ... 1 Green ... 26 Red and Green 2
C.D. Unsafe 174	77 (36.8%)	110 (63.2%)	Red ... 11 Green ... 79 Red and Green 20
Total ... 313	174 (55.6%)	139 (44.4%)	139

This supported the contention that although 1 millimetre colour targets are of undoubted value in campimetry, such small apertures are of questionable value in examinations of colour-vision outside the laboratory.

Granit supports this view when he states, "a reduction of area of the visual object, which is known to lead to disappearance of its colour with maintained brightness distribution, must do so because the "small" stimulus merely has a chance of hitting upon the common dominators.

Experimental evidence, however, shows that the recognition of a small colour light is subject to considerable individual variation.

In a series of papers published in the *Journal of Physiology* (1946), Hartridge has reported his experiments in micro-stimulation in support of his postulate that the foveal cones are frequently found grouped together into clusters by "random distribution" like the coloured starch grains on the Lumière screen.

It is not easy to see how microscopic fixation can be retained for these experiments when the eye has a constant involuntary movement over two minutes of arc of 50 per second.

These particular investigations terminated because new lanterns which were obtained had apertures varying from 1 millimetre to 0.5 millimetre and it was not possible to standardise the results.

This fact was not discovered until one group of 66 subjects

gave figures which were below the average. Only six of them were able to recognise standard green and it was found that the aperture was only 0.75 millimetre in diameter, the small difference in size being apparently of much importance.

Successive contrast

In his discussion of after-images, Edridge-Green states, "the results obtained by successive contrast are similar to those obtained by simultaneous contrast, only they are more satisfactory and decided."

When a colour is looked at for a time, the sensitivity toward that colour is lowered and the eye becomes more sensitive to the complementary. In fact, when a coloured light is concentrated upon for some minutes the complementary becomes so insistent that the original light is seen with difficulty.

If a colour-vision examination is performed slowly, successive contrast is likely to complicate the findings. It was noticed, however, that this phenomenon was less in those with normal colour vision.

Successive contrast was investigated in 415 subjects whose colour vision was abnormal. 217 (61.0 per cent.) of these were "unsafe" and 139 (39.0 per cent.) were "safe."

Using the 5 mm. aperture, standard yellow was shown rapidly after standard red, then standard green. 356 (85.8 per cent.) demonstrated the phenomenon of successive contrast.

Of the total cases, 172 were unable to recognise standard yellow after standard red, whereas only 29 failed to recognise it after green. The percentage of 50 per cent. "safe" and "unsafe" was the same in each group.

155 subjects were unable to recognise yellow after both red and green. 78 per cent. of these were "unsafe."

TABLE IX

Lantern Classification	Successive Contrast after 5 mm. aperture showing			No Successive Contrast	Total
	Red	Green	Red and Green		
Safe ...	92 (53.5%)	13 (44.8%)	34 (22.0%)	13 (22.0%)	152
Unsafe ...	80 (46.5%)	16 (45.2%)	121 (78.0%)	46 (78.0%)	263
Total ...	172	29	155	59	415

It is interesting to note among those who were "safe" according to the single lantern, the proportion (22.0 per cent.) who were unable to recognise yellow after both red and green was the same (22.0 per cent.) as those who showed no successive contrast.

During this investigation it was found that whereas the phenomenon of successive contrast is exaggerated in those with defective colour vision (Guttmann, Edridge-Green) it was not marked in the normal. A number of men were found who apparently conformed to Edridge-Green's heptachromat. They read with ease the figures in the books of test-plates "not usually seen by normal persons," and after regarding the 5 mm. aperture for some time they made no mistake whatever in naming a 1 mm. yellow or white when it followed; their successive contrast was minimal.

It was not the purpose of these tests to examine such cases further.

Simultaneous contrast

Guttmann's seventh postulate, that anomalous trichromats have a marked difference in simultaneous contrast effects as compared with the normal, is supported by Edridge-Green, who states that simultaneous contrast is not pronounced in the normal person, but the phenomenon is exaggerated to the dichromat.

TABLE X

Contrasting colours	Apparent Move
Red and Blue Green	Becomes more intense Inclines to blue
Orange and Green-blue	Inclines to red Inclines to blue
Red and Orange	Inclines to rose red Inclines to yellow
Red and Yellow	Inclines to rose red Inclines to green
Red and Yellow-green	Inclines to rose red Inclines to pure green
Yellow and Green	Inclines to orange Inclines to blue

When two spectral colours are presented simultaneously, each colour appears to be moved toward that end of the spectrum further away from the other colour. Green and rose, also red and violet appear to be exceptions.

He goes on to say, "two colours, which have not changed in the slightest degree to the normal-sighted, on being contrasted have apparently altered very considerably to the colour-blind. As an example of this, let us take a deep yellow, a bright red and bright green. To the normal sighted the yellow will be altered very little by comparison with the red or the green, but a three unit would say that the colour was green when contrasted with red, red when contrasted with green."

The effect of simultaneous contrast was investigated by showing to the candidate two colour perception units 10 inches apart at a distance of 20 ft. The following pairs of colours were shown, using the 5 mm. apertures, in any combination.

Two standard green

Two standard red

Two standard yellow

A group of 52 subjects whose colour vision was suspected who had been investigated thoroughly by other means, were examined. Of 30 who had passed successfully a single-light test, 21 made errors when shown two simultaneously.

TABLE XI

Classification according to <i>Single</i> Colour Perception Unit (5 mm.)				Classification using <i>Two</i> Colour Perception Units (5 mm.)			
Unsafe	22	Red-blind	15	Unsafe	22
		Green-blind	6				
		Red and Green-blind	1				
Safe	30			Unsafe	21
				Safe	9
Total	52			Total	52

In this group of suspects, approximately 42.3 per cent. were considered unsafe with the single light, 82.7 per cent. with the double light.

This finding indicated that the single-light lantern was not sufficiently accurate in the separation of the "dangerous" from the "non dangerous," for a candidate could not be considered safe who was unable to recognise two 5 mm. signal lights at a distance of 20 feet.

A considerable number of lanterns was made until eventually it was found that three coloured apertures, shown simultaneously, constituted the most sensitive test and a lantern was constructed which would show any combination of reds, greens, and yellows, using apertures 10 millimetres in diameter, with variable illumination.

The method of examination was as follows: the lights in the consulting room were lowered but the room was not made dark enough, or the test continued long enough, for the patient to become dark-adapted. By means of the lantern rheostat the light was increased fairly rapidly until the three apertures were distinctly seen. At this illumination the patient was asked to name the colours of the lights as they were shown to him in groups of three.

It is considered that, under these conditions, any single error in the naming of the colours shows that the man's colour vision is unsafe.

137 subjects who had made errors in reading the Ishihara plates were classified on the single-light lantern. 28 made no mistakes and were assessed "safe."

These 28 were then tested on the new lantern and 19 made errors, an increase of 13.87 per cent. in those assessed "unsafe."

TABLE XII

	Single-light Lantern	Multi-light Lantern
Safe ...	28 (20.44%)	9 (6.57%)
Unsafe	109 (79.56%)	128 (93.43%)
Total ...	137	137

The most common error was to miscall standard yellow when it was shown together with either reds or greens, or even yellows.

The next common error was to call one of a trio of reds or greens, "yellow."

If the results of the single, double, and treble light examinations are compared, it is found that the sensitivity of the test increases.

TABLE XIII

Classification of colour defectives examined by
three types of lantern

Lantern	Colour Defective Safe	Colour Defective Unsafe
Single Aperture	31.5%	68.5%
Double Aperture	17.3%	82.7%
Treble Aperture	6.57%	93.43%

It should be observed that the percentage of defectives by the double aperture is almost identical to the Ishihara.

This series of tests is evidence against the use of the single-aperture lantern and indicates the need for a fuller investigation of the anomalous trichromats hitherto considered as "safe."

Conclusions

1. This paper reports the investigation into the colour vision of 40,380 men, of whom 1,152 (3 per cent.) were safe, and 1,083 (2.5 per cent.) were unsafe.

2. Tests by colour plates, although more sensitive than tests by a single-lantern, are unreliable and differ with different series of plates.

3. The use of small apertures serves no useful purpose in the examination of colour-vision in the consulting room (Table VII).

4. The standard single-light lantern is not sensitive enough for discriminating between the "normal" and the "unsafe" (Table XII).

5. A triple-light lantern with controlled illumination is the most sensitive and reliable apparatus for demonstrating defects of the colour sense in the consulting room (Table XIII).

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A SURVEY OF ESOPHORIA AND CILIARY SPASM*

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It is with diffidence that I venture to write about ciliary spasm. Irregularities of the accommodative state seem to be on the increase and in some way connected with the post-war period, and patients are being referred in quite considerable numbers for orthoptic treatment.

I thought, therefore, that it might be interesting to illustrate several cases which have responded well to treatment.

Before discussing ciliary spasm in detail, there are one or two straightforward cases of esophoria and old accommodative squint that have received great relief as a result of treatment, and show rather clearly the different categories into which these particular forms of ocular muscle imbalance seem to be divided.

In the four groups shown in Table I we get quite a wide variation of the initial defect *i.e.*, esophoria.

TABLE I
ESOPHORIA

With normal divergence	Pseudo-esophoria	With divergence weakness
Esophoria associated with old accommodative squint		

1. *Esophoria with normal divergence.*—As is well known, this type of case is found to respond quickly to treatment and the amount of imbalance, though sometimes quite large, entirely disappears.

2. *Esophoria with divergence weakness.*—On the contrary, those in this group obtain relief from symptoms and are able to carry on their occupations successfully but owing to a divergence weakness a considerable amount of imbalance remains. Operation follows in these cases if the symptoms recur.

3. *Pseudo-esophoria.*—This is a state which is associated with

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ciliary spasm and will therefore be referred to in the second part of this paper. It is interesting to note, however, that the imbalance is very difficult to determine owing to the overaction of the ciliary muscle and the readings vary from day to day according to whether the accommodation is relaxed or working to excess. The variation can be as much as 10^{Δ} of esophoria or exophoria, and an examination on one or two consecutive days may be necessary before a decision can be made.

4. *Esophoria associated with old accommodative squint.*—In adults very severe symptoms can be experienced over a period of many years, and these can only be relieved by orthoptic treatment. The age factor is no great barrier providing binocular single vision is present, and the general health good. Dramatic changes in the

ESOPHORIA WITH NORMAL DIVERGENCE

Case 1. Age 62 years

Correction. R. + 1'25 D.sph., L. + 0'75 D.sph., Distance
R. + 3'75 D.sph., L. + 3'25 D.sph., Reading

	Before Treatment	After Treatment
Visual Acuity	R. 6/18, L. 6/18	R. 6/6, L. 6/6
B.V.A.	6/18, pt.	6/5
Worth's Lights	5	4
Maddox Rod	9^{Δ} esophoria	Orthophoric
Maddox Wing	3° esophoria	Orthophoric
Livingston Binocular Gauge	Convergence 12 cms. Accommodation 15 cms.	6 cms. 12 cms.
Synoptophore Angle ...	+ 8°	0/ + 4°
Fusion	+ 4°	0°
Adduction	8°	35°
Abduction	3°	5°
Stereoscopic Vision ...	Poor	Good

Number of Treatments 6. All tests taken with distance glasses

well-being of these patients can be noted and they state that their lives have been completely changed as a result of restored muscle balance.

In illustration of these points the following cases which fall into the several categories previously outlined have been chosen. All have been re-examined after treatment at different periods up to 2½ years and only one has had to receive a further course.

CASE NO. 1. Aged 62 years.—This patient complained of having suffered from severe headaches and giddy attacks for the past ten years, and was found to have a considerable amount of esophoria which caused her to get diplopia occasionally when tired. Being a case with normal divergence the imbalance was completely eliminated by treatment, with relief of all symptoms.

CASE NO. 2. Aged 15 years.—This child was said "to squint at meal times" and complained of headaches and diplopia when

ESOPHORIA WITH DIVERGENCE WEAKNESS

Case 2. Age 15 years

Correction. Nil

				<i>Before Treatment</i>	<i>After Treatment</i>
Visual Acuity		R. 6/4, L. 6/4	R. 6/4, L. 6/4
B.V.A.		6/4	6/4
Worth's Lights		5	4
Maddox Rod		12 ^Δ esophoria	6 ^Δ esophoria
Maddox Wing		I-11	01-3
Livingston Binocular Gauge	Convergence 14 cms. Accommodation normal	6 cms. normal
Synoptophore Angle		+ 6°	+ 1°
Fusion		+ 4°	0°
Adduction		Nil	20°
Abduction		4°	6°
Stereoscopic Vision		Too much suppression	Good

Number of Treatments 20

tired. The esophoria lessened somewhat and the symptoms disappeared, but there was evidence of divergence weakness. She has since commenced to study medicine and there is every likelihood of further treatment being needed, and possibly an operation.

CASE NO. 3. Aged 51 years.—A history of squint from early childhood was given here, and severe symptoms such as blurred vision, occasional diplopia, and acute headaches, had been experienced for the past 15 years. The prisms were removed after the first twelve treatments and at the conclusion of the second course complete relief from symptoms had been established. It will be seen, however, that the amount of esophoria remained at 12 degrees. An examination after a lapse of 2½ years found the patient quite comfortable and the measurements the same as at the last visit.

ESOPHORIA ASSOCIATED WITH OLD ACCOMMODATIVE SQUINT

Case 3. Age 51 years

Correction. R. $\frac{+1.0 \text{ D. sph.}}{+0.5 \text{ D. cyl.}}$ L. $\frac{+0.5 \text{ D. sph.}}{+0.25 \text{ D. cyl.}}$ 3 prism, B.O., E.E.
+ 2'0, added for reading

	Before Treatment	After Treatment
Visual Acuity	R. 6/5, L. 6/6	R. 6/5, L. 6/5
B.V.A.	6/5	6/5
Worth's Lights	5	4
Maddox Rod	20 Δ esophoria	12 Δ esophoria
Maddox Wing	3°-11 esophoria	Orthophoric
Livingston Binocular Gauge	Convergence 12 cms. Accommodation 23 cms.	6 cms. 18 cms.
Synoptophore Angle	+ 15°	+ 4/8°
Fusion	+ 10°	0°
Adduction	15°	30°
Abduction	5°	10°
Stereoscopic Vision	Slow	Good

Number of Treatments 24. All tests taken with distance glasses

CASE No. 4. Aged 42 years.—Here again a history of squint from four years of age with similar symptoms over a period of many years culminating finally in inability to read for more than five minutes at a time. A marked convergence insufficiency was shown at first, but later, after the removal of the prisms and a reduction of the correction, the true esophoria was revealed and treatment as for accommodative squint reduced the angle of convergence from 15° to 3° without glasses with relief of symptoms. When communicating some months later with this patient he said he had been away on holiday and had read a detective book per day.

ESOPHORIA ASSOCIATED WITH ACCOMMODATIVE SQUINT

Case 4. Age 42 years

Correction. Was wearing R. $\frac{+3.75 \text{ D sph.}}{+1.50 \text{ D cyl. } \searrow 115^{\circ}}$ L. $\frac{+3.50 \text{ D sph.}}{+1.75 \text{ D cyl. } \swarrow 65^{\circ}}$
 2 prism BOEE
 Was ordered R. $\frac{+2.0 \text{ D sph.}}{+2.0 \text{ D cyl. } \searrow 115^{\circ}}$ L. $\frac{+3.0 \text{ D sph.}}{+1.50 \text{ D cyl. } \swarrow 65^{\circ}}$

	Before Treatment	After Treatment
Visual Acuity	R. 6/6, L. 6/4	R. 6/6, L. 6/5
B.V.A.	6/24	6/6
Worth's Lights	5	4
Maddox Rod	12° esophoria	4° esophoria
Maddox Wing	4° - 6° exophoria	01° - 3° esophoria
Livingston Binocular Gauge	Convergence 12 cms. Accommodation 18 cms.	6 cms. 13 cms.
Synoptophore Angle	$+15^{\circ}$	$+3^{\circ}$
Fusion	$+12^{\circ}$	$+1^{\circ}$
Adduction	5°	40°
Abduction	5°	5°
Stereoscopic Vision	Good	Good

Number of Treatments 24. All tests taken with distance glasses.

Ciliary spasm

This condition, which can be monocular or binocular, is referred to by Air Commodore Livingston in his paper in 1934 entitled "Heterophoria in Aviation" in which he tells us of "these complicated cases showing added spasm."

These under discussion have acquired a spasm for one reason or another, each one in a slightly different way and they fit into the various categories outlined in Table II.

TABLE II
CILIARY SPASM

Spasm with heterophoria, esophoria or exophoria	Spasm without muscle imbalance	Pseudo-esophoria
Monocular or Binocular		

It is surprising to find how many variations there are, and encouraging to realise what an immensely interesting and wide field they offer to orthoptists for treatment. That these patients in some instances suffer intensely there can be absolutely no doubt. On the other hand a young person in his teens can be suffering from a marked ciliary spasm of long standing and apart from noticing the blurring, of objects in the distance, can be otherwise completely ignorant of the fact.

The symptoms vary considerably, but amongst those complained of are :—

1. Blurred vision in the distance.
2. Rapid lowering of visual acuity.
3. Pain in the centre of the eyes.
4. Frontal headaches.
5. Sickness.
6. Tensing of ocular muscles generally.

At first it was thought best to rest the ciliary muscle by application of atropine before commencing orthoptic treatment, but latterly it has proved far more satisfactory to carry out both forms of treatment simultaneously. The reason for this is that if the spasm is of long standing and therefore a very strong habit an unconscious effort will be made by the patient to overcome the

atropine, and relaxing of the muscles generally by orthoptic treatment helps to counteract this. For the same reason it has been found more helpful to give $\frac{1}{2}$ per cent. atropine b.d. rather than 1 per cent. as patients themselves in the latter case have complained of the effects of the atropine wearing off towards the end of the day with the consequent return of symptoms and renewed blurring of vision.

Unless any discomfort or irritation is experienced, with the approval of the ophthalmic surgeon, atropine is continued for as long as four to five weeks according to the severity of the case and orthoptic treatment is given twice or three times a week.

Where patients hold appointments, naturally this method of treatment necessitates sick leave, but the attempt to relax the abnormal fluctuations which occur in a ciliary spasm cannot be carried out economically by orthoptic treatment alone.

The following results will, I hope, help to illustrate the various forms of spasm treated and I will return later to the methods of treatment we have used to restore the normal accommodative state.

Two cases have been chosen to show spasm with esophoria, one being a myope and the other a hypermetrope with a very small correction. Both were young women who had had ocular symptoms for many years and finally both were compelled to give up good appointments on account of their general health being affected.

CASE NO. 5. Aged 26 years.—This patient had suffered for six years from severe headaches and a gradual onset of blurred vision accompanied by attacks of vomiting lasting for several days.

Her visual acuity deteriorated starting in July, 1939, as R. 6/9 L. 6/5. December, 1941, R. 6/9 L. 6/12. January, 1944, R. 6/36 L. 6/36.

Maddox Rod 8 Δ esophoria and Maddox Wing 11—13 esophoria in glasses.

The binocular visual acuity at the commencement of orthoptic treatment in August, 1944, was 6/24 with glasses. This was restored to 6/9pt. after 24 treatments with complete relief of symptoms there being no further attacks of vomiting after the spasm had started to relax.

However, after a period of 2 $\frac{1}{2}$ years the patient felt a slight tendency to return to the old habit of ocular tension and on examination it was found that the B.V.A. was 6/12. However, after a further course of treatment this was rectified. The cause of this relapse was thought to be due to a serious shock with the expected temporary setback to general health.

CILIARY SPASM WITH ESOPHORIA

Case 5. Age 24 years

Correction, R. $\frac{-5.00 \text{ D sph.}}{-1.00 \text{ D cyl. } \Delta 90^\circ}$ L. $\frac{-4.50 \text{ D sph.}}{-1.00 \text{ D cyl. } \angle 90^\circ}$

	Before Treatment	After Treatment
Visual Acuity	R. 6/24, L. 6/24	R. 6/12, L. 6/12
B.V.A.	6/24	6/9 pt.
Worth's Lights	5	4
Maddox Rod	8 Δ esophoria	6 Δ esophoria
Maddox Wing	11 $^\circ$ -13 esophoria	3 $^\circ$ -5 esophoria
Livingston Binocular Gauge	Convergence 10 cms. Accommodation 16 cms.	6 cms. 13 cms.
Synoptophore	+ 5 $^\circ$	0/+ 2 $^\circ$
Fusion	0 $^\circ$	0 $^\circ$
Adduction	30 $^\circ$	30 $^\circ$
Abduction	5 $^\circ$	7 $^\circ$
Stereoscopic Vision ...	Good	Good

Number of Treatments 36. All tests taken with glasses.

CASE NO. 6. Aged 26 years.—The history here revealed a tendency towards headaches for several years and the patient said she was often told at school that "her eyes were looking queer." Because she never felt very comfortable when reading, on volunteering for national service at 18 years she joined the Women's Land Army to avoid close work.

After several years' service she unfortunately met with an accident and fractured the base of her skull, and this precipitated acute ocular symptoms with occasional diplopia at the end of the day.

She was subsequently referred to an orthoptic clinic and found to have full ocular movements but 17 $^\circ$ of esophoria.

She received 12 orthoptic treatments without relief and no reduction in the imbalance, although every effort was made to help her and her co-operation was excellent.

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The surgeon decided that operation was not indicated at that stage but if no improvement occurred during the next 18 months he would reconsider the case.

In the meantime the patient was referred to me for investigation. I discussed the condition very fully with my colleague and we both agreed that there was some further problem yet to be discovered. This finally proved to be a ciliary spasm and by the time I saw the patient the vision had started to deteriorate to 6/18, either eye and binocularly.

Atropine was given together with treatment and the vision improved and the symptoms almost disappeared, the convergence angle being reduced from 20 degrees to 0 degrees as the result of the relaxing of the spasm.

Atropine was discontinued after a period of four weeks and very great care was taken to ensure that the patient completely understood how to relax her eyes and the importance of guarding against the possible return of the spasm.

CILIARY SPASM WITH ESOPHORIA

Case 6. Age 26 years

Correction Nil

			<i>Before Treatment</i>	<i>After Treatment</i>
Visual Acuity	R. 6/18, L. 6/18	R. 6/5, L. 6/5
B.V.A.	6/18	6/5
Worth's Lights	5	4
Maddox Rod	22° esophoria	9° esophoria
Maddox Wing	5°-11 esophoria	3°-9 esophoria
Livingston Binocular Gauge	Convergence 6 cms. Accommodation 6 cms.	6 cms. 12 cms.
Synoptophore	+10 20 after atropine 0	0 + 4
Fusion	+10° after atropine 0	0
Adduction	20	35
Abduction	3	5
Stereoscopic Vision	Poor	Good

Number of Treatments 36.

She suffered acutely when learning to adduct and converge and frequently had attacks of vomiting after treatment.

The importance of helping the patient to persevere through these difficult phases of the treatment cannot be too strongly stressed and providing the health is normal in every other respect, orthoptic treatment should not be discontinued until they are overcome. In almost every case the same difficulties occur when trying to develop convergence without the return of the overaction of the ciliary muscle.

The effort to converge causes a very real disturbance which results either in nausea or in actual vomiting and one learns the necessity to watch the colour of one's patient and limit the time and method of treatment accordingly.

CILIARY SPASM WITH EXOPHORIA

Case 7. Age 17 years

Correction. R. $-8^{\circ}0$ D. Sph. L. $-8^{\circ}0$ D. Sph.
 $-0^{\circ}50$ D. Cyl. $\times 60^{\circ}$

	<i>Before Treatment</i>	<i>After Treatment</i>
Visual Acuity with glasses	R. 6/18, L. 6/18	R: 6/9, L. 6/9
Without glasses ...	R. 6/36, L. 6/36	R. 6/18, L. 6/18
B.V.A. with glasses ...	6/9 pt.	6/6 pt.
Without glasses ...	6/36	6/18 pt.
Worth's Lights ...	5 crossed	4
Maddox Rod ...	4 Δ exophoria	1 Δ exophoria
Maddox Wing ...	18 Δ exophoria	6 $^{\circ}$ - 8 exophoria
Livingston ...	Convergence 14 cms.	6 cms.
Binocular Gauge ...	Accommodation 6 cms.	12 cms.
Synoptophore ...	-5° later -10°	0/ - 2°
Fusion ...	-4°	2° -
Adduction ...	5°	40°
Abduction ...	8°	10°
Stereoscopic Vision ...	Good	Good

Number of Treatments 30. All tests taken with glasses.

CASE NO. 7. Aged 17 years.—Here the spasm was slight, in fact only just commencing, and no symptoms at all were complained of in spite of a quite considerable amount of exophoria.

However, no attempt was made at first to reduce the exophoria but rather to relax the muscles, thereby revealing a further amount of imbalance, but at the same time restoring the visual acuity to normal. After reaching this stage the treatment proceeded as in the usual way for exophoria, observing closely for any return of the spasm on adduction and convergence. No atropine was used in this instance.

CASE NO. 8. Age 51 years.—We now come to a most interesting type of spasm, *i.e.*, that without any muscle imbalance of which only two cases have been referred for treatment, one having a binocular spasm and one a monocular. The first patient had been ill for some months and had suffered recently from alopecia areata.

CILIARY SPASM WITHOUT MUSCLE IMBALANCE

Case 8. Age 51 years

Correction. R. +1.75 D. Sph. L. +1.75 D. Sph.

	<i>Before Treatment</i>	<i>After Treatment</i>
Visual Acuity	R. 6/60, L. 6/60	R. 6/6, L. 6/6
B.V.A.	6/60	6/5
Worth's Lights	4	4
Maddox Rod	2 ⁺ esophoria	orthophoric
Maddox Wing	orthophoric	orthophoric
Livingston Binocular Gauge	Convergence 16 cms. Accommodation 10 cms.	6 cms. 19 cms.
Synoptophore Angle ...	0°	0°
Fusion	0°	0°
Adduction	20°	30°
Abduction	4°	6°
Stereoscopic Vision ...	Poor	Good

Number of Treatments 15. All tests taken without glasses.

She had had a long period of convalescence and came complaining of "going blind" saying that her vision had failed during the last four days. She also said that she had been compelled to hold her book nearer and nearer to her eyes.

As will be seen she could only see 6/60 E.E. and binocularly and, as was to be expected, was in a highly emotional state. She had rather poor stereoscopic vision, considerable suppression on the synoptophore, but otherwise showed no imbalance. The real trouble was detected when testing the accommodation on the Livingston Binocular Gauge which with and without glasses was 10 cms. instead of 19-20 cms. at the age of 51.

Atropine was prescribed and on account of the age of the patient, this was carried out under the personal supervision of the ophthalmic surgeon. It was discontinued after 12 days as the visual acuity had recovered to 6/9 and orthoptic treatment was given to strengthen the stereoscopic vision and relax the remaining spasm and thus restore visual acuity to 6/5 and the accommodation to 19 cms.

The patient made a rapid recovery to good health and has not had any return of the trouble.

CASE NO. 9. Age 14½ years.—The second case in this group which shows a monocular spasm, was quite different in that there were no symptoms complained of, merely the fact that the vision was "poor" in one eye.

Here we find the spasm tending to lessen over a period of time, rather than increase, as in most instances.

From the orthoptic point of view there was little to account for the condition except that the stereoscopic vision was very poorly developed indeed and there was marked suppression of the right eye with poor convergence.

The variation in the refraction, however, is of great interest as in 1944, the R.E. vision was corrected from 6/18 to 6/5 with R.E. -2.0 D. Sph. L. plane; 1945, the correction was reduced to -1.0 D. Sph.; 1946, the correction appeared to be the same, the vision being improved from 6/36 to 6/6 in glasses.

In all these previous instances, atropine had been given for three days prior to refraction.

In August, 1946, it was decided to continue atropine for fourteen days and the correction then accepted was R. + 0.5 D. Sph., L. + 0.5 D. Sph.

Atropine was continued for a further fourteen days, glasses were discarded and the right eye vision improved from 6/36 to 6/6 after twelve treatments.

This patient has now returned to school and will be seen at half

term to check the vision as we do not know as yet how well she will be able to stand up to close work.

It would seem that some patients suffering from ciliary spasm accept a myopic correction and to a certain extent are comfortable and, for a time, able to see better.

Sooner or later, however, the spasm increases, the symptoms become more acute and finally the glasses are no help.

CILIARY SPASM WITHOUT MUSCLE IMBALANCE

Case 9, Age 14½ years

Correction. April 1944, R. -2.0 D. Sph. Left Plane.

August 1946, R. +0.50 D. Sph., L. +0.50 D. Sph. After atropine for 14 days.

	<i>Before Treatment</i>	<i>After Treatment</i>
Visual Acuity with glasses	R. 6/5, L. 6/5	—
Without glasses ...	R. 6/36. L. 6/5	R. 6/6, L. 6/5
B.V.A. without glasses ...	—	6/5
Worth's Lights ...	4	4
Maddox Rod ...	orthophoric	orthophoric
Maddox Wing ...	orthophoric	orthophoric
Livingston Binocular Gauge	Convergence 6 cms. Accommodation 8 cms.	6 cms. 13 cms.
Synoptophore ...	0°	0°
Fusion ...	0°	0°
Adduction ...	4°	20°
Abduction ...	2°	6°
Stereoscopic Vision ...	Very poor	Improved

Number of Treatments 12. To continue. All tests taken without glasses.

CASE NO. 10. Age 15½ years.—This condition often calls for atropine, as usually there is exophoria present although esophoric readings on the Maddox Rod and Wing may be recorded.

In this particular case it will be seen that the reading on the

Maddox Rod was orthophoric, the Wing 5-9 esophoric and Worth's Lights 5 homonymous.

After atropine the Maddox Rod showed 7^{Δ} exophoria, the Wing orthophoria; and the synoptophore varied from $+5$ degrees to -12 .

Another case not illustrated here showed 10^{Δ} of exophoria and -5° on the synoptophore on one day and on the following day 8^{Δ} of esophoria and $+10/15$ on the synoptophore. These abnormal fluctuations lasted until atropine was given and the exophoria revealed was 10^{Δ} .

PSEUDO-ESOPHORIA

Case 10. Age $15\frac{1}{2}$ years

Correction. R. $+4.25$ D. Sph. L. $+5.5$ D. Sph.
 -1.25 D. Cyl. -1.75 D. Cyl.

	Before Treatment	After Treatment
Visual Acuity with glasses	R. 6/6 pt. L. 6/6 pt.	R. 6/5, L. 6/5
Without glasses ...	R. 6/12 L. 6/12	R. 6/9, L. 6/9
B.V.A. with glasses ...	6/5	6/4
Without glasses ...	6/9 pt.	6/6
Worth's Lights ...	5 homonymous	4
Maddox Rod ...	Orthophoric. After atropine 7^{Δ} exophoria	7^{Δ} exophoria
Maddox Wing ...	5° - esophoria. After atropine orthophoric	orthophoric
Livingston Binocular Gauge	Convergence 6 cms. Accommodation 10 cms.	6 cms. 12 cms.
Synoptophore ...	$+2^{\circ}$ - 10 after atropine	0°
Fusion ...	0°	0°
Adduction...	10°	28°
Abduction ...	4°	10°
Stereoscopic Vision ...	Good	Good

Number of Treatments 18. Still continuing. All tests taken with glasses.

Diagnosis

The history reveals repeated efforts to clear the print when reading finally resulting in the necessity to hold the book nearer and nearer to the eyes.

Sickness and giddiness accompany these extreme efforts causing an acute emotional state on account of the loss of vision.

General muscle tension, not only of the ocular muscles, adds to the aggravation.

In spasm the ciliary muscle is contracted causing excess accommodation. This state gives rise to the following indications.

1. Variable visual acuity with and without glasses.
2. Blurred vision in the distance.
3. Distant vision sometimes cleared by concave lenses.
4. Near point focus.
5. All usual signs of myopia.
6. Evidence on the synoptophore, *i.e.*, blurring, suppression, variable angle.
7. Readings on all tests vary from day to day.
8. The reading on the Livingston Binocular Gauge when testing accommodation are usually far in excess of the normal.
9. The resolving of all these states after the application of atropine.

Treatment

There are very few new ideas, if any, in the treatment of these cases, but, as previously mentioned, there is a considerable emotional element present and marked muscle tension generally, and the whole basis of the treatment is relaxation.

It might be argued that atropine does this for the patient but in many cases the brain is so accustomed to the bad habit that it makes every effort to overcome the effect of the atropine, and it is here that the orthoptist can be of greatest assistance in helping the patient to know what his eyes are doing, and by learning to relax, gradually place them in a position of rest.

In the final stages of treatment a very high standard is aimed at in the effort to restore the accommodative mechanism to normal and in doing so the smallest slides are used, and finally when teaching adduction the smallest Maddox fusion slide has to be mastered to at least 25-30 degrees.

Comparison between these figures and those required for a cure in convergence insufficiency would be unfavourable, and it must be remembered that the tendency for the ciliary muscle to overact again requires long and patient handling before a cure is obtained.

Summary

In conclusion, I think it is wise to assume that we are, as yet, only on the threshold of this extremely interesting application of orthoptic treatment and much will be revealed as time goes on.

The most important factor to stress is the necessity of testing the accommodation both binocularly and monocularly in patients of all ages and types excluding those actually suffering from strabismus.

CONTACT LENSES IN EXCELSIS*

BY

ALLAN H. BRIGGS

LINCOLN

IN 1938 I was consulted by a young man aged 22 years, who was anxious to join the Civil Air Guard. He had been rejected on several occasions at various centres owing to the fact that he was rather highly myopic. He was extremely interested in flying, and very keen to learn, but his myopia had proved an insuperable obstacle. He was anxious to know of any form of treatment, operative or otherwise, which might overcome his difficulty; and the only possible course I had to suggest was contact lenses. He was very willing to bear the expense and to try the experiment and I accordingly placed him in touch with the contact lens centre. His visual acuity was: R.V. c—8.0 D.sph. and—1.0 D.cyl. axis $170^{\circ} = 6/6$. L.V. c—4.0 D.sph. and—2.0 D.cyl. axis $170^{\circ} = 6/5$.

I heard later that he had been successfully fitted with contact lenses and had presented himself for medical examination again, without divulging that he was wearing contact lenses, and had been passed without their presence being suspected.

I lost touch with him during the war period, but was able to establish contact with him again recently after he had been demobilised. I was anxious to learn what had transpired, and I do not think I can do better than quote his letter to me: "... Briefly I enlisted in the R.A.F. in a ground trade in February, 1940, but transferred to flying duties during the following year. I had the usual medical examination, and was posted to Canada and America for training as a pilot. My lenses were not detected either at this medical Board, or at two subsequent Boards I had in America and upon my return to England. The American Board carried out

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by American doctors, was if anything, more thorough than those conducted by R.A.F. medical officers.

"After I returned from the States I was given some operational training and then posted to a Bomber Squadron as a pilot and completed quite a number of sorties over Germany. I was flying as Captain of Halifaxes at this time. Things went well until January, 1944, when my aircraft came into contact with some ironmongery over Berlin and as a result we had to abandon the thing by parachute some time later. I was extremely lucky and managed to get into France, there joining up with the Resistance people. After a few weeks with them things got a bit hot and I had to get out of France which I did by climbing the Pyr nees and getting into Spain. After a short period there I got back to England through the good offices of the Embassy in Madrid.

"Upon my return to England I had to undergo a further medical board—this was usual after one had become a casualty, but the results were the same as before. My medical category throughout was A.1b, and eyes 6/6. 6/6. The remainder of my service was as Captain of Transport aircraft (Liberators and Dakotas), first from this country to India, N. Africa, etc., and later I was posted to Australia for similar duties. I stayed there just over a year returning to England in April last, and from then until the end of my service on 1st of January this year (1947) I was employed as a test-pilot. My medical examination revealed the same result! I had no difficulty or trouble in the air irrespective of height or manœuvres, nor has service in tropical climates caused any discomfort. I managed to avoid dust storms, or the story might have been different—dust and grit is most unpleasant!

"I had two pairs of contact lenses, and always carried a spare pair with me; fortunately I never had cause to resort to the spare pair. None of my colleagues ever had the slightest clue that I wore these things; and the methods by which I contrived to wash and clean them at night, and replace them in the morning, are rather amusing. Nevertheless I always managed to carry out the operations without a great deal of difficulty. During the day I seldom had cause to remove them. The only occasions (fortunately rare) were when I had been a bit careless in putting them in, and had got a lash under them. I usually managed to find a quiet corner to remedy this trouble.

"I wear them for up to 17 or 18 hours a day, but normally for 16 hours. On two occasions only, when circumstances pressed a bit I wore them for periods of 30 and 36 hours respectively but I must admit my eyes were a bit 'tired' after this, and were bloodshot.

"I usually go to the contact lens centre about once a year for a check—the last time I went no change of power was necessary.

I am due to go any time now, and it may be that some slight change of power will be needed, as the 6/6 line was not quite so clear with one eye as it had been on previous examination, on my last examination by the R.A.F.

"I am not altogether severing all connections with flying, and I shall join an auxiliary Air Force squadron, or do some private flying. Consequently I still desire the secret to be kept and I know you will treat the whole matter with the confidence that it needs in the circumstances. Of course I have no objection to you using my experiences, provided my name does not appear connected therewith. Incidentally, even my wife does not know about them—I was married in August last year."

(The patient omits to mention in his letter that he was decorated for "courage, determination, and devotion to duty").

It seems to me that there are certain important matters which arise out of this history.

(1) It seems clear that all the duties of an R.A.F. pilot can be carried out efficiently while wearing contact lenses. Even allowing for the fact that this may be an exceptionally successful case, and perhaps an exceptionally resolute patient, the position suggests that the visual acuity regulations for the services ought to be revised to show to what extent contact lenses will be permitted. If they give a satisfactory standard of visual acuity and can be worn for a reasonable length of time, it is difficult to see why they should not be allowed. If their use is permitted, it might permit many disappointed men who are otherwise physically fit, to have a second chance.

(2) It is manifest that contact lenses are not likely to be detected during a routine medical examination, even during one in which the estimation of visual acuity is regarded as of great importance. Ought any steps to be taken with regard to this? How far is it justifiable to aid and abet a patient in procuring contact lenses, knowing they are likely to be used to defraud a medical examiner, and how far is such a subterfuge unfair?

(3) The patient's letter demonstrates something of the future possibilities of contact lenses, both as regards their value as a visual aid and also as regards their difficulty of detection.

In conclusion, I should like to pay tribute to the courtesy and co-operation of the staff of the Contact Lens Centre, London, to whom all credit for the successful result in this case should be given.

ANNOTATION

Spare copies of the earlier volumes of the Transactions of the
Ophthalmological Society of the United Kingdom.

We are informed by the Council of the Society that the printers have a large number of the earlier volumes of Transactions in stock. We understand that of the first 10 years (1880-89) there are more than 100 volumes. The younger members of the fraternity should embrace the opportunity of completing their sets. Only those who in the past have endeavoured to make good gaps in a long set of periodicals will know the difficulties and appreciate the excitement when a volume that at first sight seemed unobtainable comes in. The writer was once speaking on this subject to the late Mr. Treacher Collins and was told by him of the struggle he had to acquire the first volume of the Ophthalmic Hospital Reports, and of the high price he had to pay for it. Very early in our career we were lucky in purchasing vols. 2-7 of the Ophthalmic Hospital Reports at a reasonable price. We never saw a copy of the first vol. for sale but when our senior colleague retired from hospital he gave us his copies of vols. 1 and 2 which he had acquired for a shilling the pair at a sale of books at the beginning of the present century! In such a way do values and prices vary.

If the younger members of the Society do not rise to the bait the Council, in our opinion, would be well advised to consider either selling at a nominal figure or making a present of the earlier sets to such University Libraries among the allied countries of Europe as suffered damage and loss in the last world war. There must be many in Russia, France, Holland, Belgium, Norway and Denmark that would be glad to accept such an offer.

[The name and address of the printers is Headley Bros., The Invicta Press, Ashford, Kent. London office, 109, Kingsway, W.C.2. Telephone, Holborn 3309].

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM. ANNUAL CONGRESS

THE 67th Annual Congress of the Ophthalmological Society of the United Kingdom was held in the Department of Zoology, Glasgow University, on March 27, 28 and 29. The President of the Society, Professor A. J. Ballantyne was in the Chair. He delivered an address of welcome to the members of the Society and to visitors

from abroad, in particular, Dr. P. Merigot de Treigny (Paris), Dr. E. Godtfredsen (Denmark), Dr. J. Van Caneyt (Belgium), Professor W. H. Melanouski, Madame Schiss-Wertheimer and Madame Dallon. One hundred and eleven members attended the Congress.

The Presidential address, "De Senectute" was a clever and entertaining survey of the organic and functional changes that come to most of us whom the relentless march of time carries on to old age. Professor Ballantyne gave a general view of the tissue changes that occur with advancing years and in particular the familiar ocular disorders of cataract and presbyopia. The President gave useful advice about the explanation of these disorders to patients and the help this gave in their adjustment to such defects. He discussed the limitations of treatment and the possibility that new therapeutic agents might in the future postpone senile degenerative changes.

The subject of the Discussion was "Rhino-logy in Relation to Ophthalmology." The openers were Dr. J. Marshall, who dealt with the ophthalmological aspects of the subject, Mr. G. H. Howells described the part played by the Ear, Nose and Throat surgeon and Dr. R. McWhirter showed some admirable radiographs to demonstrate the importance of these in the diagnosis of inflammatory disorders, neoplasms and injuries of the orbit and adjacent structures. The openers had agreed not to discuss nasal sinusitis as a possible cause of retro-bulbar neuritis and intra-ocular inflammation.

The trend of the discussion was in favour of referring cases of orbital inflammation to the rhinologist. With his co-operation sulphonamide and penicillin therapy is carried on. It is preferable to drain into the nose an abscess originating from an infected sinus. If drainage is indicated through the orbit then it is best to do this in a plane between the orbital periosteum and the bone and not to open the orbital fascia.

The value of radiotherapy in the treatment of malignant neoplasms which have originated in the accessory nasal sinuses and have invaded the orbit was described and this treatment is preferred to surgical removal, particularly in the case of malignant neoplasms of the antrum.

For the exploration and removal of neoplasms deep in the orbit the relative merits of the trans-frontal approach and the lateral orbital access through Krönlein's operation were briefly discussed. There is no doubt that the former is indicated in cases when it is likely or possible that the neoplasm has extended beyond the confines of the orbital cavity and the latter is well justified when it is certain that the neoplasm is probably innocent, such as a cavernous haemangioma, neuro-fibromatosis and a cyst.

Opinion was more in favour of Toti's operation, dacryocystorhinotomy, or a modification of this for naso-lacrimal duct obstruction than of West's intra-nasal operation.

Orbital and ocular injuries as a result of nasal sinus operations were mentioned. Such calamities as penetration of the eye, orbital cellulitis, total ophthalmoplegia and division of the optic nerve are among these.

The subject rhinology in relation to ophthalmology afforded much material for discussion.

Other papers of interest were keenly discussed. These were:—"Induction of an Experimental Tumour of the Lens," Professor Ida Mann. "An Unusual form of Retinal Detachment (? cystic) seen in Children," Mr. F. A. Juler. "A Classification of Epiphora, with remarks on diagnosis and treatment," Dr. H. M. Traquair. "Some Aspects of Lid and Socket Repair," Mr. J. Scott Tough. "Bilateral Retinal Detachment Associated with Choroidal Cyst; a clinical and pathological report," Dr. J. Pendleton White and Dr. I. C. Michaelson. "Corneal Blood Staining," Professor A. Loewenstein. "Reiter's Disease," Mr. R. Lindsay Rea. "The Relation between Sjögren's Disease the Plummer-Vinson Syndrome, and Ariboflavinosis," Dr. E. Godtfredsen. "Observations on Holes at the Optic Disc," Mr. H. Neame. "Self-Inflicted Eye Injuries," Dr. L. B. Somerville-Large. "Vision During Glancing Movements of the Eyes," Dr. G. H. Bell and Dr. J. B. de V. Weir. "Vascular Disturbances in the Eye following Concussion Injuries," Dr. A. M. Wright Thomson. "Cysticercus Cellulosae of the Eye," Professor W. H. Melanowski. "Schnabel's Cavernous Atrophy," Mr. Eugene Wolff. "Some Aspects of Disease Affecting the Retinal Veins," Dr. A. J. Ballantyne and Dr. I. C. Michaelson.

A Clinical Meeting was held at the Glasgow Eye Infirmary. Twenty-seven interesting cases were shown and discussed.

The Annual Dinner of the Society was held in the Hall of the Royal Faculty of Physicians and Surgeons. Sir Andrew Davidson proposed the toast of the Society and the President replied. In a pleasing and witty speech Dr. S. Spence Meighan proposed the toast of the Guests. Dr. P. Merigot de Treigny and Professor Geoffrey B. Fleming replied to this.

Sir Stewart Duke-Elder's speech about the President was an admirable survey of Professor Ballantyne's qualities as a man and an ophthalmologist of international repute.

At the Annual General Meeting the Secretary read the Annual Report of the Society's activities. It is evident from the Treasurer's report that the Society's finances are in a sound and flourishing state.

A Trade exhibition of Ophthalmological Instruments and Appliances was held during the Congress.

BOOK NOTICES

The Surgery of Repair Injury and Burns. By D. N. MATTHEWS, MA., M.CH., F.R.C.S. Pp. 371, illustrations 198. Blackwell Scientific Publications, Ltd., Oxford. Price, 45/-.

The author has based the material of this book on his experiences as a surgeon in the R.A.F.V.R. where he has treated a large number of war injuries and burns, and has followed up these cases. He has omitted orthopaedic work, but inserted an account of the routine treatment of perforating wounds of the abdomen and thorax. The book deals mainly with the common war injuries which come within the province of the plastic surgeon. He describes the technique he favours. The chapter on the treatment of burns and shock is particularly well written. The book is well illustrated, many of the illustrations are in colour.

Chapter XIV deals very briefly with a few plastic operations for the eyelids and contracted socket. This chapter is not well done and contains a number of errors, some serious. The description of ptosis pays too little attention to the cause and the degree of ptosis and the manner in which these facts affect the treatment. The author does not comment on the undesirability of placing a load on the superior rectus, neither does he state in his description of passing fascia lata strips between the lid and the frontalis muscle for ptosis of what he calls neurogenic origin that any operation to raise the lid in III nerve palsy (ophthalmoplegia externa) condemns the patient to diplopia and so should not be done. His advice about placing a "pressure pad" over the eye after a ptosis operation is inviting a severe corneal abrasion. His histological knowledge is seriously at fault when he describes the tarsus as composed of "cartilage."

The best ptosis operations such as advancement and partial resection of the levator palpebrae superioris and Blascovics's operation are not mentioned.

It is difficult to understand why dacryocystorhinostomy is considered a plastic operation. It is evident from the author's description that he has never seen the operation done properly. The incision he advises is much too far forward, and with this it is little wonder that he is embarrassed by haemorrhage from the angular artery and recommends that "it is wise to spend some time in identifying and ligating it before proceeding with the removal of the bone." There are many important technical details omitted, and the lacrimal retractor shown in Fig. 154 is so crude that it would damage the eye during operation.

We cannot agree that the raw area for paramedian tarsorrhaphy need be as long as 1 cm., nor that a Wolfe graft has poor vitality.

Stent mould pressed into the socket in socket reconstruction gives a deep irregular shaped socket with an uneven surface. It is better in the reviewer's opinion to use an acrylic mould made of the shape of the normal conjunctival sac after excision of the eye, in fact the shape of a prosthesis, and to attach to this the graft for insertion into the socket.

The book is well produced, the paper and illustrations are good. It is a useful and practical contribution to traumatic and plastic surgery.

Klinische und Erbbiologische Untersuchungen ueber die Heredoataxien. By T SJÖGREN. Acta Psych. et Neurol. Supp. 27. Copenhagen. 1943.

A survey of the literature concerning the hereditary ataxias shows that much difficulty is experienced in separating all the diverse clinical types. Numerous transitional forms, *e.g.*, between Strümpel's spastic degeneration and the Charcot-Marie neuromuscular atrophy, have been described. A recent and very thorough study of the subject has been made by Dr. T. Sjögren, whose work on the genetics of diseases of the central nervous system is already well known. The new monograph describes cases and their families discovered through the Swedish hospitals. All cases of Friedreich's ataxia, of Marie's ataxia and of other similar ataxias were collected. The material is fully analysed from the clinical point of view, details of family histories and geographical distribution of cases are given. There are numerous illustrations and tables. Altogether, 95 male and 93 female cases in 118 families are reported.

Dr. Sjögren divides his material into four groups and one subgroup. Groups I and IA contain cases of Friedreich's ataxia—those in IA were less typical than those in I. The whole group contained 101 cases in 64 families. The frequency of this disease in the general population is estimated to be 0.0035 per cent. Fifteen per cent. of the cases were mentally defective. The mean age of onset in the typical Friedreich cases was 12 years and in the less typical cases, 15 years. A strong tendency was found for the age of onset to be constant in different affected members of the same family. This may indicate that, even within this selected group, there is more than one type of disease. All parents were unaffected. In six families the patients were first cousins and in three families they were more distantly related. This high consanguinity incidence coupled with the estimated familial incidence in sibships of about one quarter makes it highly probable that Friedreich's ataxia is recessively inherited. The possibility that there may be more than one allelic gene accounting for differences in onset age in different families is not discussed in the monograph nor is the material critically examined for evidence of partial sex linkage.

Group II contains 12 cases of transitional type in 12 families with mean age of onset of 50 years.

Sixty-four typical cases of Marie's ataxia in 35 families are classed in Group III. The mean age of onset here was 34 years, and in each family one parent was affected. There seems to be little doubt that the condition is dominant but, as with Friedreich's ataxia, affected members in the same family tend to have similar onset ages. The incidence of Marie's ataxia in the Swedish general population is estimated at 0.0014 per cent.

Dr. Sjögren's Group IV contains 11 cases of a mild hereditary type of ataxia, present from birth and lacking progressive tendency. Here the cause is almost certainly a dominant gene.

The method of combining genetical and clinical data before attempting to sort out different disease types is coming more and more into general use. The three types of data, clinical and pathological, statistical—age of onset and rate of progress of the disease—and family history material are all considered by Sjögren in the grouping of his cases. Though some transitional cases are still not fully accounted for, the method clearly enables great strides to be made towards the goal of defining the main types of nervous diseases and ascertaining the causal mechanisms behind them.

Les yeux et la vision des vertebres. ROCHON DUVIGNEAUD. Pp. 718, 500 illustrations. Paris: Masson et Cie. 1943.

It is a heartening thing to find so beautifully produced and so comprehensive a monograph appearing from Paris in 1943. Despite innumerable difficulties of paper shortage, permits, restrictions and set-backs of all kinds the doyen of French comparative ophthalmology has crowned his life's work with this outstanding publication and both he and Masson are to be congratulated. The death of the artist, M. Bessin, almost in the act of completing the superb illustrations, is a tragedy, since the completed work is as great a monument to his skill and industry as it is to its author's erudition and painstaking research.

The treatise deals with the gross and minute anatomy of the eyes of all the subdivisions of the vertebrates. It is written from the point of view of the human anatomist and ophthalmologist rather than the phylogenist and therefore the first 150 pages deal in detail with the structure of the human eye. Thereafter we are led from palaeontology, through vertebrate embryology to comparative anatomy of a most comprehensive type. The 500 beautiful illustrations form a collection only equalled by that of Gordon Walls and definitely surpassing the older monograph of Ovio. Throughout, the stress is on structure and accuracy of measurement and many of the data concerning the dimensions of the various vertebrate eyes are only available here. The work is essentially the result of painstaking

personal observation. Its insistence on structure, largely to the exclusion of speculations as to function, makes it a most valuable companion volume to that of Walls, with its phylogenetic and adaptational descriptions of function. Every ophthalmic library in the world will be incomplete without a copy.

Die Erkrankungen des Augenhintergrundes. By Dr. ADALBERT FUCHS. Franz Deuticke, Vienna. 1943.

In 1943 Adalbert Fuchs published a book in Vienna on Diseases of the Fundus, copies of which have now come to this country. The book is essentially a pathological treatise with an atlas of drawings of fundus diseases and drawings of the corresponding histological sections. It is divided in two parts. The first is a simple factual description of the commoner pathological conditions affecting the retina, the choroid or optic nerve; it is didactic in nature, without discussion and without references to the literature. The second part is a comprehensive atlas in which each case is fully reported and each picture described; many of the cases have already appeared in the literature. Some of the drawings are good, and some of them not convincing; for example, both the painting of the fundus and that of the microscopical section illustrating solitary tubercle of the nerve-head are lacking in sufficient detail to convey an adequate impression of the condition. Now that the technique of histological photography has reached so high a standard, it would seem that little is to be gained by semi-diagrammatic and somewhat artificial paintings which can convey little to the advanced student or the critical reader. At the same time the book, considering the date of its publication, is beautifully produced.

CORRESPONDENCE

ASTIGMATISM IN INDIANS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—Duke-Elder quotes Nordenson (1883) as proving the existence of physiological astigmatism, averaging 0.50 — 0.75 D., and being “with the rule” (the axis of least curvature not more than 30° from the horizontal) in 89 per cent. of a series of cases.

Refraction of Indian troops suggested that this rule did not apply to them, and so I have analysed 1449 consecutive cases of astigmatism refracted in my department. This includes all refractions; not only those for whom glasses were prescribed. The Indians were all adult males, the British included some military families.

BRITISH

Age	Total	All cases		Below 1.0 dioptre	
		With the rule	Against the rule	With	Against
Below 20 ...	49	28	21	8	8
20-29 ...	273	187	86	86	62
30-39 ...	135	83	52	39	36
40 and above	84	35	49	26	43
Totals ...	541	333	208	159	149

INDIAN

Below 20 ...	51	19	32	17	16
20-29 ...	367	151	216	91	156
30-39 ...	269	65	204	44	149
40 and above	221	49	172	36	119
Totals ...	908	284	624	188	440

While the British cases follow the "rule" much less closely than expected, the Indian actually show a majority against it. The total figures shown as percentages are:—

	Below 1.0 D.		All cases	
	With	Against	With	Against
British	51.5	48.5	61.6	38.4
Indian	30.0	70.0	31.3	68.7

With increasing age there is the usual increase of cases against the rule: the only keratoconus had 8 D. of astigmatism with the rule. These are two facts commonly quoted in support of the contention that physiological astigmatism is due to lid pressure on the globe.

The only factor which I can suggest as responsible for this inversion of the rule in Indians is trachoma, which is found in a majority of the soldiers seen in this part of India, and often in severe form. This is offered solely as a suggested explanation: I find no reference to the condition in the text-books available, and have no access to the literature.

I wish to thank the Commanding Officer, British Military Hospital, Delhi for leave to publish this note.

Yours faithfully,

R. A. D. CRAWFORD.

Major, R.A.M.C., Ophthalmic Specialist

AGRAM, BANGALORE,
INDIA COMMAND.

THE "NORMAL" IN THE SYNOPTOPHORE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—Referring to the paper by M. M. Lewis in your December issue and to N. Cridland's comments in the February issue, I would suggest that the latter's statement that "... I do not think that there is, on the face of it, reason to suppose that the synoptophore and the Maddox rod measure different qualities of heterophoria" should not pass without comment.

I am not clear as to the meaning of the term "*qualities of heterophoria*" but, with regard to the degree of heterophoria that will be revealed, surely when the two eyes are dissociated so that the visual mechanism finds itself in an unusual state of balance, or imbalance, to deal with which it has no safe guide from its normal experience, it is to be expected that the position taken up by the eyes will fluctuate round about a mean position which may be definitely affected by the kind of apparatus that has been used to secure dissociation. This is to be expected especially when testing at near, when the whole mechanism is in a dynamic state and by suspending fusion we have eliminated the incentive for the governing innervation of the complex group of innervations that are in operation. I think that many practitioners will agree that this expectation is borne out in practice: for the results obtained,

especially in near testing, with the Maddox rod method do not in general agree with those obtained with the synoptophore.

Yours faithfully,

H. H. EMSLEY.

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March 13, 1947.

REMOVAL OF THE WRONG EYE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—I am grateful to Dr. Ascher for his letter on my paper on "Removal of the wrong eye." At the same time I do not follow his inference that the paper suggests that a case of this kind occurred in Elschmig's clinic either before or after 1908. Dr. Ascher's remarks about the meaning of "Soll" are, of course, correct, but seem to me to be irrelevant. I made no attempt at a literal translation as I thought that clumsy English would result and the free rendering I gave of the sentence in question conveys no implication of the kind Dr. Ascher indicates.

I have no information that such a case ever occurred in Elschmig's clinic but I am assured that Elschmig used to impress the dangers of this accident on his students, which suggests that he had heard of at least one case. The significance of his foot-note is clear: it means that between writing his text and correcting the proofs—otherwise why a foot-note?—he had learned of another case. I was careful to write "within his knowledge." The case may have occurred anywhere, possibly outside Austria altogether.

Cutting the eyelashes is only another method of marking the eye, though perhaps a better one than the use of adhesive plaster. The important point is that, for one reason or another, methods of marking the eye have proved unreliable.

I have for many years given up cutting the lashes in eye operations except sometimes a few at the lateral and medial ends of the upper lid. I doubt whether any advantage is gained by cutting even these.

Yours faithfully,

H. M. TRAQUAIR.

16, MANOR PLACE,
EDINBURGH, 3.

April 10, 1947.

FOUND—PATHOLOGICAL SPECIMENS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—A few days ago I was informed by the police that a package has been found in Lincoln, comprising a bottle containing two human eyes and a smaller bottle containing what appeared to be a crystalline lens, complete in its capsule.

These specimens appear to have been removed by an ophthalmic surgeon recently. How they came to be abandoned in Lincoln is at present a complete mystery and it seems possible that they may have been stolen from an ophthalmic surgeon's car and abandoned by the thief on discovering their nature. We have made local enquiries but have so far failed to discover any clue as to the owner of these specimens and I wonder if you would give publicity to the matter from your columns in the hope that the ophthalmic surgeon concerned may chance to hear of the matter in this way.

We should be very grateful if the rightful owner will get in touch with me, and on supplying particulars I should be pleased to arrange for the specimens to be handed over to him. It seems more than likely that these specimens were of some special value and we are anxious to restore them to their owner if possible.

Yours faithfully,

ALLAN H. BRIGGS.

3, LINDUM ROAD,
LINCOLN.
April 8, 1947.

THE USE OF UNIT CELLS IN THE LISTER
MORTON OPHTHALMOSCOPE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I have noticed that if I use unit $1\frac{1}{2}$ dry batteries in my Lister Morton Ophthalmoscope, they often run down prematurely.

The following paragraphs explain the reason and give the remedy for this occurrence, which I am sure many users of the instrument must have also experienced.

The correct dry battery to use is, of course, the Ever Ready 1829, and this gives no trouble in use.

However, it is not so easily procurable as the standard $1\frac{1}{2}$ volt unit cell, and the latter is, I think, most commonly used.

One of the features of the Lister Morton Ophthalmoscope made by Theodore Hamblin, Ltd., is a retaining ring of metal which is inserted at the upper end of the battery handle.

This ring retains the battery, when the head of the instrument is removed from the handle for insertion into the instrument case provided, and is a useful feature of the ophthalmoscope. Unfortunately the $1\frac{1}{2}$ volt unit cells are so manufactured that the zinc casing, which is the negative pole, is exposed at the upper end of the cardboard insulating envelope, and can make contact with the retaining ring of the battery handle.

This causes a short circuit of the lower cell in the battery handle, and only happens when the handle is detached from the head of the instrument.

The remedy is either to depress the battery a little in its cardboard container and roll the edge of the cardboard over the upper rim of the zinc casing of the cell, or to glue a thin ring of cardboard as an insulator under the metal ring in the ophthalmoscope handle.

The correct battery for the ophthalmoscope, the Ever Ready 1829, is a two cell, 3 volt battery, enclosed in a single cardboard envelope the ends of which are rolled over, and so prevent the short circuit described when using two single cells.

Yours faithfully,

P. T. LEES.

7, ALBANY AVENUE,
BLACKPOOL.

OBITUARY

IAN STEWART MCGREGOR

OPHTHALMOLOGY could ill afford the death of Dr. Ian Stewart McGregor on January 23 at the age of 43 years, when already he had shown great capacity as clinician, surgeon, teacher and original investigator. He graduated M.B. CH.B. at the University of Glasgow in 1927 and thereafter filled various resident appointments. His attainments were all the more remarkable in that his interests in ophthalmology began only nine years ago, previous to which he was in general practice on the Island of Bute. This experience in general medicine, however, so broadened his outlook and sharpened his judgment that he learned quickly and fastidiously from his colleagues and within a brief period was appointed Clinical Assistant at the Glasgow Eye Infirmary and Assistant Ophthalmic Surgeon to the Ophthalmic Institution of the Glasgow Royal Infirmary and obtained the Diploma of Ophthalmic Medicine and Surgery granted by the Royal Colleges in England. At the outbreak of war he was mobilised as a squadron leader in the R.A.F.V.R. where he served for two years. His release was requested in 1941 to fill a vacancy as Visiting Surgeon to the Ophthalmic Institution. He also acted



IAN STEWART MCGREGOR

as Senior Assistant to the University Department of Ophthalmology. Despite the great demands of hospital practice in the war years he became a Fellow of the Royal Faculty of Physicians and Surgeons and of the Royal College of Surgeons in Edinburgh. He graduated M.D. in 1943, the subject of his thesis being the "effect upon the eyes of methyl alcohol poisoning." Self critical he published only after his precise staking of the known boundaries of ophthalmology. His publications included the following important papers:—

Orbital cellulitis from gas producing organisms. *Brit. Med. Jl.*, 1, 292-293, 1942.

Reticulin content and prognosis in malignant melanoma of uvea. *Arch. Ophthalm.*, 30, 291-297, 1943 (with Hill, J.).

Study of histopathological changes in retina and late changes in visual field in acute methyl alcohol poisoning. *Brit. Jl. Ophthalm.*, 27, 523-543, 1943.

Quinine blindness. *Lancet*, 2, 566-567, 1944 (with A. Loewenstein).

Bilateral partial ectasia of nerve head with peripapillary ectasia. *Brit. Jl. Ophthalm.*, 28, 618-622, 1944.

Macular coloboma with bilateral grouped pigmentation of retina. *Brit. Jl. Ophthalm.*, 29, 132-136, 1945.

Segmental movement of pupil. *Brit. Med. Jl.*, 1, 629-630, 1945.

Cyclic oculomotor palsy. *Jl. Neurol. Neurosurg. and Psychiat.*, 8, 22-23, 1945.

His scientific enthusiasm was tempered by the wider necessities. He was a dependable colleague, and his knowledge of the known limits and an imagination for the stretch beyond made his opinion on a case or problem sound and stimulating. This was often freshened by his piquancy of phrase. His patients were particularly fond of him, his keen mind and natural compassion rarely leading him astray in that proper blending of assessment and charity which they require. Deeply interested in literature and in the countryside he was a good example of what these, combined with an experience of general practice can do to perfect the interests and quality of a speciality. He was unmarried, and our very sincere sympathies go to his mother on her great loss.

NOTES

Honneur

MR. COLVILLE MUIRHEAD has been elected an Hon. Member of the Société belge d'ophtalmologie.

Appointment Dr. P. H. BEATTIE has been appointed ophthalmic surgeon to the Norfolk and Norwich Hospital.

National Society for the Prevention of Blindness. THE National Society for the Prevention of Blindness announces the appointment of Dr. Franklin M. Foote to the position of Executive Director succeeding Mrs. Eleanor Brown Merrill who is retiring. Mrs. Merrill has been associated with the Society for more than twenty-five years and has served as the Executive Director for the past eight years. She formerly held the positions of Associate Director and Secretary.

Dr. Foote joined the Society's staff as Medical Director in 1946. He was formerly District Health Officer of the Kips Bay-Yorkville Health District of the New York City Health Department. Prior to that, he was Chief of the Division of Local Health Administration, Connecticut State Department of Health. Dr. Foote is Assistant Professor of Public Health and Preventive Medicine at Cornell University Medical College. Dr. Foote holds degrees of B.S., M.D., and Dr. P.H. from Yale University. During World War II, he served as a Major in the Medical Corps of the United States Army.

White Oak Hospital, Swanley, Kent. 1944-45 DATA on admissions during 1944-45 are incomplete owing to the evacuation of the Hospital during part of each of these two years. For 1946 there were 222 admissions distributed as shown:

Blepharitis	79
Phlyctenular ophthalmia	109
Interstitial keratitis	4
Recurrent styes	16
Others	14
						222

In addition the Ophthalmia Neonatorum Unit which is still housed at White Oak, admitted 163 babies and 30 nursing mothers. Eight babies and eight mothers required re-admission.

IIIrd Congress of Pan-American Ophthalmology THE Congress will be held in Habana from February 22 to 28, 1948. Dr. Tomas R. Yanes is President of the Local Committee and will be assisted by members of the Sociedad Cubana de Oftalmologia.

Corrigenda IN Dr. Inglis Pollock's letter, p. 123, the following corrections should be made. Line 14, 303 should be 503. Line 15, Anglicum should be Anglicam and translation should be translatum. Line 16, pages should be folios. Line 17, 68 should be 136. Line 20, MMS. should be MS.

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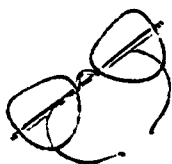
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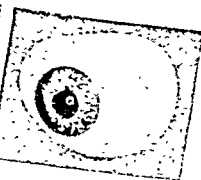
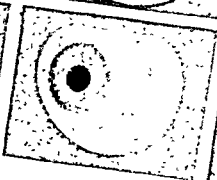
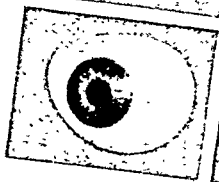
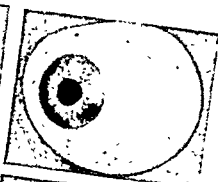
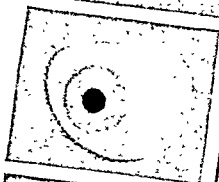
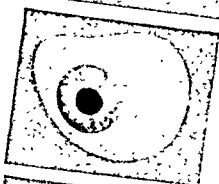
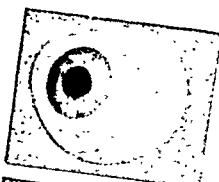


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BINOCULAR VISION * †

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ALTHOUGH binocular vision has not so far been made the subject of a Middlemore Lecture—certain allied disorders of vision have been discussed by previous lecturers. It is interesting to find that Mr. Fulford Eales—in 1920—deplored the failure of the Education Authorities to deal adequately with cases of visual deficiency, and particularly with those suffering from squint and amblyopia—while Mr. Haycraft, from his experience in examining recruits in 1943, found the incidence of amblyopia so high, that he too deplored the fact that so many apparently healthy eyes should grow up practically blind. Moreover, he regarded it as “a slur on the care which we oculists devote to our young helpless patients.” The interval between the two lectures was that of a complete generation—and it seems to be in keeping with the

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progressive spirit of Richard Middlemore that we should meet this challenge by a consideration of the physiological factors which maintain binocular vision, and an inquiry as to how we can employ this knowledge—to improve the treatment of cases of abnormal binocular vision.

In presenting my thoughts to you I shall undoubtedly repeat certain familiar facts, but I hope to present them to you in fresh perspective, and to include in them an account of recent observations. The one-eyed man is perhaps not now regarded as an oddity, but he is certainly debarred from many interesting activities, while perfect binocular vision is required for so many occupations in the skilled industrial world of the immediate future, that its lack may determine the fate of a man. The high standards of binocular vision required by the Royal Air Force in this last war led to a mass-enrolment of orthoptic trainers, and the work which they accomplished under the guidance of Air Commodore Livingston in restoring binocular vision to pilots—has I think removed the last barriers which existed between ophthalmic surgeons and orthoptists as to the usefulness of orthoptic training. Indeed, it repeats the challenge to ophthalmic surgeons henceforth to make their proper contribution towards the art of orthoptic training—in order that it shall not run the danger of becoming merely a technical exercise.

The common disorders of binocular vision are well known to you—viz. primary amblyopia—and the allied condition of suppression; strabismus—both paralytic and concomitant—and heterophoria.

Paralytic squint, from its sudden onset and its embarrassing clinical symptoms of double vision and false projection, has been recognised by physicians for many years and has been correctly attributed to a pathological disorder of the cranial nerves or nuclei supplying one or other of the extra-ocular muscles. Concomitant strabismus on the other hand was first regarded as a naturally occurring malposition of one eye—and Dieffenbach in 1840 was the first to attempt its surgical correction. Nevertheless, as long ago as 1743 Buffin had suggested that it was due to inequality of vision between the two eyes—while over a century later Priestley Smith ascribed it to a disturbance of those cortical nervous centres which govern binocular vision.

Up to the beginning of this century—the treatment of squint consisted in a correction of the errors of refraction and cosmetic operation. Claud Worth (1902) was the first to inquire into the significance of the amblyopia which is so often present in a squinting eye—and to treat it by occlusion of the good eye—followed by simple binocular fusion exercises on the amblyoscope.

He believed that a fusion centre existed in the brain which was essential to binocular vision, and was absent or lacking in function in cases of concomitant strabismus. The realisation that a faulty state of binocular vision, *viz.*, heterophoria, can exist without an obvious malposition of the eyes—came later still.

Now if, in the light of our present knowledge, we consider the factors which contribute to the maintenance of perfect binocular vision in man, we find that they are (1) The overlapping of visual fields (2) A strongly developed sense of fusion—and (3) The dominance of macular over peripheral vision.

1. *The Overlap in Visual fields.*—Let us first consider the origin of the overlap in visual fields. Most of us are sufficiently familiar with the phylogenetic development of animals, to know that the eyes were first placed laterally—and then assumed a frontal position. We must also bear in mind the fact that in order of development, conjugate movements of the eyes preceded convergence.

What is known as the nasal half of the retina must originally have been occupied with the receipt of visual stimuli arising *behind* the animal—and with the assumption of the frontal position of the eye—this half of the retina is still occupied with stimuli arising on the outer side of the body. In the first case the reaction of the animal as a whole will be to move away from the object behind it, and in the second case—the animal will be stimulated to make divergent movements of the eyes.

The temporal halves of the retinae, on the other hand, are concerned with the reception of stimuli arising *in front of* the animal. The eyes are mutually attracted instead of being distracted—and there is every incentive for the eyes to converge in an attempt to locate the object more precisely. When the visual axes of the two eyes meet—there is a considerable overlap of the visual fields—which must increase the dominance of the temporal halves of the retinae and strengthen visual attention in the binocular area. A re-arrangement of the optic nerve fibres occurs first in the cats, when instead of there being a total decussation of fibres—the semidecussation with which we are familiar is instituted. It is noteworthy that this anatomical linkage appears to subserve conjugate vision; while the non-decussation of fibres from the temporal halves of the retinae favours binocular vision.

Another interesting anatomical fact is that the power of *convergence* only occurs in animals which possess maculae—*i.e.*, the higher monkeys and man. It is associated with a great development of the brain. Not only are there visual sensory areas in the occipital cortex, but a prefrontal cortical area which is associated

with the acquisition of a wide range of conjugate movement and with convergence. In fact, Kennard (1940) found that destruction of one entire frontal lobe in monkeys had the same effect as destruction of one occipital area, in that the animal did not appear to see objects in a contralateral field of vision. In other words though the sensory stimulus was undoubtedly being received by the animals, appropriate mental reaction was stopped. The *localisation* of an image in a visual field is not then purely a sensory phenomenon but is a sensory-motor response. Holmes has reported two cases in which impairment of the parietal lobe in a patient caused defective spatial orientation—although the visual fields were unimpaired and visual acuity was fair. Riddoch (1935) has pointed out that visual disorientation can occur when the association pathways from the occipital cortex to other parts of the brain are interrupted, the nodal point appearing to be in the region of the supramarginal and angular gyri in the parietal lobe.

This new anatomical evidence is against the theory held by so many ophthalmologists that spatial localisation arises purely from the position of the stimuli which fall on the retinae, or from any exactly recorded pattern of them in the visual sensory cortex.

It is probably the correct conclusion to say that the overlap of visual fields lays the foundation of binocular vision, by concentrating visual attention, so that an object can be located correctly, and that the added powers of convergence and accommodation—serve to make the location precise. The mere overlap of fields does not guarantee the fusion of two images into one. For this a fusion sense appears to be necessary—and is intimately bound up with the sense of projection.

Fusion Sense.—Paralytic squint is the most dramatic instance of a breakdown of fusion. It arises from a motor defect in the extra-ocular muscles, which disturbs the normal position of the visual axes. An attempt has been made to explain the accompanying false projection, also on the basis of a failure of *motor* innervation. It was said to be due to the fact that "When an ocular muscle is weak the increased innervation of the opposing muscle gives an impression of a greater movement of the eye than has really taken place; thus the mind receives the idea that the object is placed farther to one side than it really is, and in an attempt to touch it, the finger may go beyond it." Even so eminent a physiologist as Gowers stated that "We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the *innervation we give to the ocular muscles.*"

While such an argument might conceivably be true for paralytic

squint, in which defective orientation is partly overcome by secondary deviation of the other eye, and by alteration of head-posture, it is contrary to the facts to interpret all abnormalities of binocular vision as a failure of motor innervation. In concomitant squint the visual axes retain the same relationship to one another over a wide range of conjugate movement—and a *sensory* disturbance occurs in the form of amblyopia or suppression in the majority of cases. Worth explained the onset of suppression as being due to the absence of a fusion centre in the brain—but there is no physiological or anatomical evidence for the existence of such a centre. There is obviously a *sensory-motor* balance in binocular vision—and Franklin (1942) has also emphasised the idea that the functions of fusion, projection and stereopsis depend on this balance—rather than on visual sensations which arise from retinal stimuli alone.

He points out that before the development of the eyes the movements of the body were guided by the impressions received by the lateral sense organs, and that in course of time the eyes took on the same function as the sense organs. We have further evidence of this in the fact that no bird will fly in a fog, because it loses its directional sense, and that a blindfolded pigeon, when loosed from an aeroplane, fails to fly, and drops like a stone.

In animals with laterally placed eyes in which the binocular fields do not overlap, *e.g.*, herbivores and fishes, the animal shows that it can see an object ahead by moving up to it and seizing it. In the carnivores, which have frontally placed eyes—there is a definite correlation between vision and the use of the forelimbs, in the catching and killing of fast-moving prey. The mode of life adopted by the primates, of swinging by hand from branch to branch—requires an exact sense of distance—and projection of an object in space, and we find the anatomical response—of specialisation of the brain, convergence, accommodation and macular vision to which I have already referred.

Thus, in animals, in the fundamental association of body action with visual stimuli, each forelimb is correlated with vision in the *contra*-lateral field. In man, the intimate association of body movement with visual stimuli is well shown in the lack of general co-ordination which often accompanies paralytic strabismus, and is also seen in certain cases of concomitant strabismus when the patient attempts to rely on the amblyopic eye alone.

Franklin has emphasised the fact that fusion and projection in binocular vision are based on *bitemporal* retinal representation—*i.e.*, on the overlapping of nasal fields, but that they are anticipatory in character—and comparable to potential movements.

Let us now consider the third factor which influences binocular vision—*viz.*, *macular dominance*.

Birds with laterally placed eyes, whose visual axes are set at an angle of 120° or more, have a fovea, so that it would appear that the primary function of the fovea cannot be one of fusion. Nocturnal birds of prey, with frontally placed eyes, have two foveae in each eye.

The macular fibres in man are distributed over an area which extends for approximately 20° in a horizontal direction on either

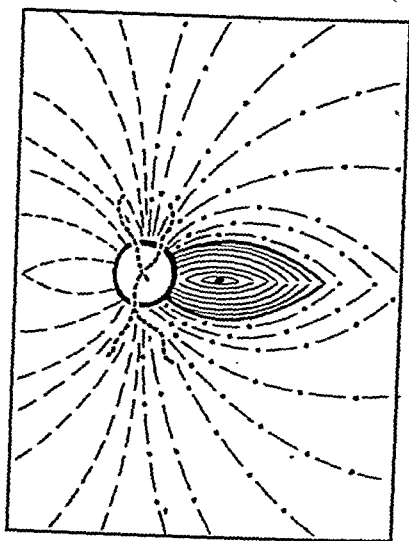


FIG. 1.

The course of the visual fibres of the retina.

side of the macula and embraces the blind spot on the nasal side (Fig. 1). Vertically the area extends for about 3° above and below the macula. Visual acuity within this central area rises from 6/60 at the periphery to 6/6 at the fovea (Fig. 2).

On account of the superiority which this visual acuity and pattern vision has conferred on man—we are apt to consider that it dominates over the more primitive visual function of fusion. It is important to remember in this respect, that bitemporal retinal representation persists throughout the central area of the retina—and the overlap of the visual fields must be as shown in Figs. 3 and 4. One would expect to find, as is indeed the case, that fusion is very strong in the paramacular area, and it decreases peripherally.

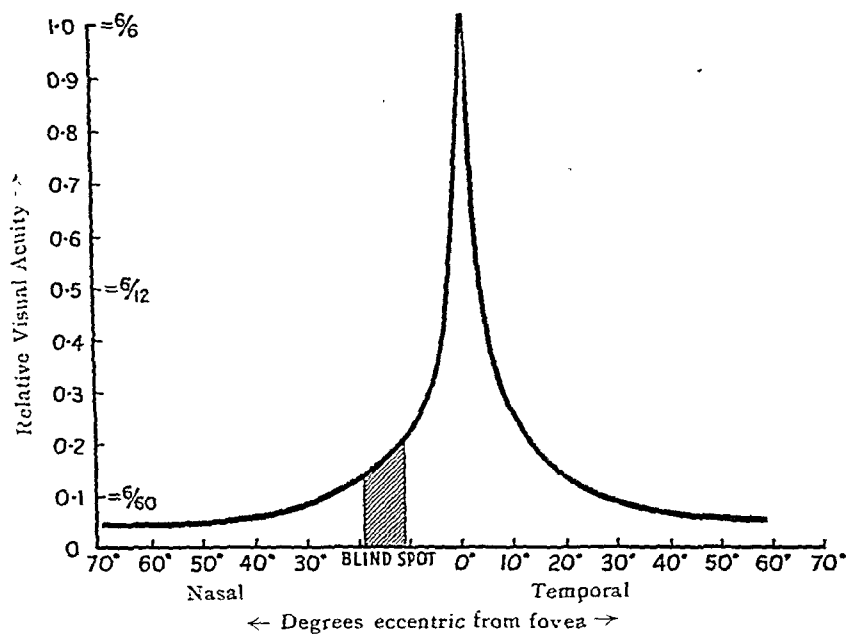


FIG. 2.

The regional variation of visual acuity (after Wertheim).

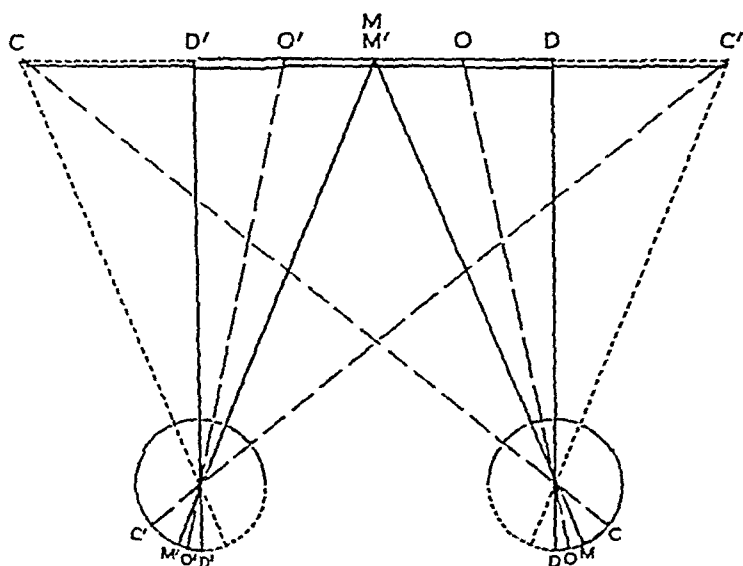


FIG. 4.

Overlap of visual fields in binocular fixation (central area).



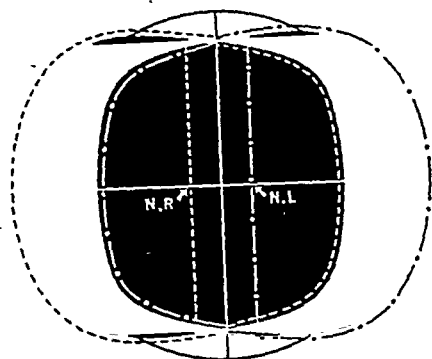


FIG. 3.

The overlap of visual fields in man.

□ Binocular vision. ■ Monocular vision.

n.r. = nasal limit for "central field," right eye. n.l. = nasal limit for "central field," left eye.

The maintenance of perfect binocular vision must depend on a bilateral harmony within the central area, of the two mechanisms of pattern vision and fusion. This probably occurs only in a minority of individuals, since the possibilities of the breakdown of so fine an adjustment are so many. It has in fact been said (Bielchowsky, 1937) that only about 20 per cent. of individuals with good binocular vision are really orthophoric, and recent observers have found evidence of a continual competition between foveal vision and the fusional processes occurring in the area immediately outside the fovea.

In certain pathological conditions there is a surprising maintenance of binocular vision; *e.g.*, subjects suffering from retinitis pigmentosa, in which the function of the peripheral retina is entirely lacking, can still maintain binocular vision when only a small central field remains and their visual acuity is very low. Conversely subjects with bilateral macular degeneration or disease also may retain some degree of fusion. A study of binocular vision in conditions of partial loss of visual field, would, I think, elucidate the functional balance between the peripheral and central areas of the retina, still further.

In a series of tests on another aspect of this problem, which have been made recently at this hospital, we have found that good binocular vision persists in the dark adapted eye—even when the illumination of the test object is so low as to favour rod, rather than cone, vision. The subjective angle tends to become convergent; there is a general tendency to giddiness, and colours are

suppressed. Some degree of stereopsis persists even when visual acuity is greatly diminished,

The *physiological* abnormalities of binocular vision (see Table 1) may be tabulated as shown. We find all degrees of variation, from a complete competition between the eyes, as in alternating strabismus, down to total suppression of central vision in one eye. Thus at each end of the scale—vision becomes uniocular rather than binocular. In the intermediate grades some attempt

TABLE I
Physiological Abnormalities of Binocular Vision

1. Alternating strabismus	...	Uniocular vision
2. Congenital nystagmus	...	Usually some degree of binocular vision
3. Heterophoria	}	Abnormal binocular vision due to rivalry within the "central" area of the retina
Low degrees of concomitant strabismus		
False associated fixation		
4. Large degrees of concomitant strabismus	}	Abnormal function in both "central" and peripheral retina
(a) with, and (b) without suppression		
5. Advanced suppression with loss of central fixation	...	Peripheral function only in affected eye; binocular vision poor or absent

at binocular vision is made. In by far the majority of cases this is accomplished by the partial suppression of pattern vision—which has been, abundantly proved to be a mental process of ignoring the image from one eye. In others, although visual acuity is full, foveal fixation gives way in order that extra-foveal fusion can be maintained; or competition is avoided by establishment of a false macula—in the condition we recognise as false associated fixation.

When the central area of one eye is very defective—abnormalities of orientation occur. The onset or non-occurrence of strabismus is, I believe, determined at its onset by the initial level of visual acuity in the eye affected. By referring again to the curve for visual acuity (Fig. 2) we are reminded that the central area includes the blind spot, so that "the margin of escape" is only 10° on the nasal side of the macula—and any failure in this region will favour the development of a convergent strabismus.

You will notice that I have so far avoided making any reference to the subject of retinal correspondence. It has long been considered that stereopsis was due to the stimulation of corresponding points on the retina, and to an exact reinterpretation of such stimuli in the visual sensory cortex. I have already shown that this is not true in the case of the primitive fusional mechanisms—and recent work has shown that it is too precise a theory to be applied even to foveal fixation.

McAllister (1905), Dodge (1907) and Clark (1936) have shown that in normal binocular vision an area of $1-1.5^\circ$ from the fovea

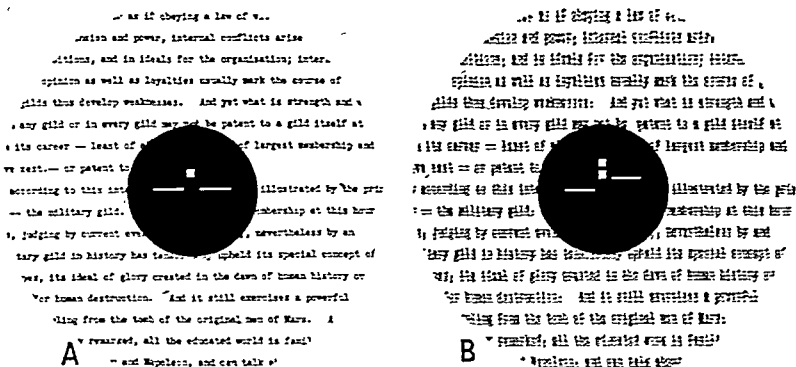


FIG. 5.

Diagram showing the doubling of a centrally fixated object (1° square) by displacement of peripheral images (print), A, superimposed, B, displaced (Burian, 1939).

can be used for fixation—and with a disparity of $2-4^\circ$ horizontally and 2° vertically in either direction an object will still be reported as single. Burian (1939) made a further discovery that peripheral retinal stimuli, if displaced, by as small an interval as 0.25° , will break the fusion of images situated on corresponding areas of the macular region, and no matter how hard the observer tries he is unable to maintain central fusion in the presence of displaced peripheral stimuli (Fig. 5). Burian made these tests within an area of 12° round about the macula. Stimulation of smaller areas of $\frac{1}{2}-1^\circ$ at a distance of 12° from the fovea caused noticeable fusional movements of the eyes. Burian concluded from his observations that there must be a large number of individuals who will never be able to fuse central and paracentral images simultaneously; some in fact maintain a rapid small alternation.

He later (1941) examined 75 cases of concomitant strabismus with less than 12° strabismus and full visual acuity. He found that the eyes performed small fusional movements in order to maintain

binocular vision, and that patients who had no foveal suppression, showed a small degree of suppression in the paramacular area. Paramacular fusion could only be maintained at the expense of foveal fixation being out of alignment. The exceptions were (a) alternation, due to suppression of all the images of each retina

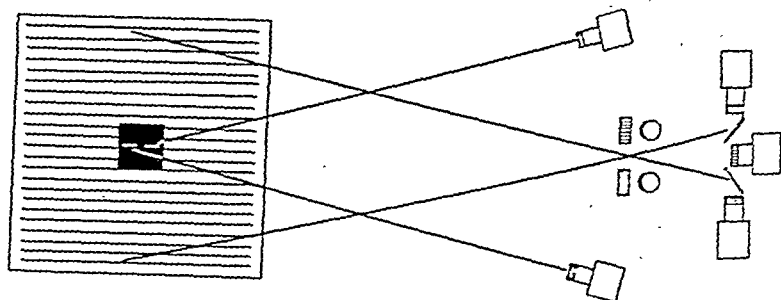


FIG. 6.

Peripheral fusion (of superimposed line pattern) and foveal test lines on a level. Images seen as above, i.e., unchanged by (a) Patient with normal binocular vision. (b) Patient with strabismus with anomalous correspondence adapted to the angle of squint (Burian, 1941).

in turn; (b) The presence of a false macula, which supported all the attributes of binocular vision except stereopsis (Fig. 6). His methods of experiment were both accurate and ingenious.

A comparison of the average number of eye-movements made per second, while the eyes "fixed" isolated points and points in context. (Clark), 1936.

(By kind permission of Professor Clark, *Amer. Inst. Psychol.*, 1939)

	Horizontal component		Vertical component	
	R. E.	L. E.	R. E.	L. E.
Isolated point A ...	7.55	7.01	4.11	6.20
Point A in context (i.e. viewed in a picture)	6.88	7.59	3.88	6.39
Isolated point B ...	6.98	6.66	4.28	5.53
Point B in context	7.08	6.55	3.83	5.44

FIG. 7.

The improvement of the camera has made it possible to make objective records of the movements of the eyes—rather than having to rely entirely on subjective phenomena.

Judd (1907) was the first to photograph the movements of the eyes in convergence and divergence. He found that as they viewed two fixed points in space, the eyes of some subjects made conjugate movements prior to convergence, while others moved one eye faster than the other. In short there was no exactitude of movement in fixation. The same observation was made by Clark (1936) who from an analysis of over 3,000 photographic records found that the quality and quantity of the movements was the same for stereoscopic vision, as in the fixation of an isolated point (Fig. 7). The eyes made an average of 8 movements per second, but one eye moved more often than the other (Fig. 8). Clark

Average number of movements per second made by the eyes
fixing points in a Stereogram (Clark).

(By kind permission of Professor Clark, *Amer. Inst. Psychol.*, 1939)

Fixating					Fixating				
Near Point			Far Point		Near Point			Far Point	
Subject	R.	L.	R.	L.	Subject	R.	L.	R.	L.
1	5.9	9.9	10.2	13.8	9	5.3	8.1	3.3	7.0
2	5.1	5.8	6.0	6.2	10	4.1	7.0	11.9	9.2
3	11.8	13.7	15.9	14.1	11	9.0	6.8	8.2	7.2
4	7.7	9.2	9.1	9.9	12	5.8	2.4	3.1	3.8
5	7.1	9.8	8.9	12.0	13	8.3	6.9	8.8	10.2
6	9.8	7.2	8.7	10.9	14	11.1	9.9	10.0	9.1
7	9.0	11.7	5.9	5.8	15	5.2	7.1	5.1	5.2
8	7.9	8.3	6.2	10.8	16	—	—	8.8	11.8

FIG. 8.

concluded that clear visual perception can only occur at the movement of fixation—and in the interval visual acuity must be diminished. Obviously then, visual perception must constantly be both binocular and monocular, and shift from one eye to another. He suggests that the highest grade of binocular vision must therefore depend on the organisation and unification of minimal changes—rather than on the stimulation of specific geometrical or corresponding points on the retina. He believes that stereopsis must be made up of a rapid comparison of uniocular impressions, and in this he is supported by Litinski (1939) who found that in 72.5 per cent. of normal subjects which he examined, the uniocular perception of solidity was equal to the binocular perception. Verhoeff too supports the theory that binocular vision occurs by uniocular replacement—rather than by simultaneous vision of the two eyes. He believes too, that while one eye is occupied with the observations of one portion of space, the other eye is suppressed in this region only—so that the two eyes divide between each other the entire field of vision in an ever-changing mosaic of small uniocular part-fields. Verhoeff's conclusions were based on subjective phenomena—but the objective experiments of Burian and Clark go far to substantiate them. In an analysis of amblyopia which I made earlier this year (1946) I arrived at the same conclusion, *viz.*, that binocular vision depends on some form of physiological replacement. It is certainly the only theory which explains all the abnormalities of binocular vision, particularly those which arise from functional disorders of the central area of the retina. Thus Hering's theory which states that in binocular vision the image is projected along the visual axis of a hypothetical cyclopiian eye—midway between the two eyes—appears obsolete. The elaboration of this theory to explain stereopsis as due to the simultaneous stimulation of corresponding and disparate points of the retina also appears to me to be impossible. I was surprised to find that the latest definition of stereoscopic vision which has been given to orthoptic students is "the ability to fuse images falling on *slightly* disparate points of the retina." It is difficult to reconcile this with any theory of binocular vision.

In conclusion, I shall attempt to apply these newer concepts of binocular vision to the practice of orthoptics.

The young child resembles the animal in its development, and its early years are spent in learning to correlate its body reactions with visual stimuli.

I cannot resist quoting from Richard Middlemore's "Treatise on diseases of the eye," certain passages in which he lays stress on the very early onset of strabismus (1835).

In early life, children are sometimes placed very injudiciously in their cradle, or they are, perhaps, very generally carried in one and the same position, so that the light gains more frequent and complete access to one organ than to the other, in consequence of which, that eye which is most familiarized with the influence of the luminous rays, is more readily stimulated by them, and the other is rendered, in a corresponding degree, the more insensible to them. Again, sometimes the infant's cap is ornamented with a long and partly coloured border, so that it readily attracts the child's attention, and he is continually turning the eyes towards it, as he lies in the cradle. In the same way a small spot, naevus, or tumour upon the nose, may produce squinting by disposing the child to look at it very intently, and he is inclined to make great and repeated efforts to turn the eyes towards the object of attention, because it is not distinctly seen. Of these two causes of infantile strabismus, the former will cause strabismus divergens, and the latter strabismus convergens.

7.—Imitation is certainly a cause of strabismus; parents will often tell their medical attendant that their child did not squint when he left home for school, but, on his return, the strabismus was complete; and the child will probably mention that many of his school-fellows also squinted, because they were frequently mocking or imitating each other. No cause of strabismus is more satisfactorily made out than this.

8.—Sometimes one of the eyes happens to be inflamed in early life, and it is carefully bound up, until its visual powers become impaired from prolonged repose from its natural actions and the long absence of its natural stimulus; such an eye will be liable to be affected with strabismus, unless, which is very improbable, its retina should again recover its sensibility. In other instances both eyes may, perhaps, be slightly inflamed, and the surgeon may tell the parents of the child to protect them from the influence of bright light, and they will bind them up, as they conceive, very carefully, in order to fulfil what they presume to be the Doctor's wish the better, and in this way the patient will be totally deprived of his sight during the continuance of the ophthalmia, at least, according to the parent's intention; but children do not like this deprivation, and therefore so change the situation of the bandage, as to see either upwards or downwards, and, in this way, acquire a habit of squinting. Many of the worst cases of strabismus of both eyes, which it has fallen to my lot to notice, have been so caused.

It is certainly a pity to lose the early years as a time for training. The sense of fusion can only be properly developed if the two eyes are used equally. Visual acuity should be brought to a maximum by ordinary glasses at as early an age as possible—and occlusion should not be left, as it so often is in the hospital child, until the age of 5 (Fig. 9). It can be employed much earlier—or perhaps a better method is the use of atropine drops in the good eye, in order to bring about fixation in the squinting eye. I have never known this method to cause false associated fixation to develop—and it has the advantage that it can be started in infancy—and that the child continues to exercise some degree of binocular vision—whereas occlusion cut out any stimulus to fusion. There is a good deal of evidence that the amblyopic eye supplies a spatial background to the good eye, and this must be the explanation as to why quite a number of adult amblyopes have straight eyes and a certain degree of fusion.

The next stage of fusion training is not usually started until about the age of 6, because the child cannot understand the synoptophore and has not sufficient mental application. I think more use might be made of the toy games in the interim.

The development of visual acuity in the young child, following treatment. (Campbell, 1946)

TREATMENT UNDER 5 YEARS					TREATMENT OVER 5 YEARS	
Group A			Group B		Group C	
Vision	Before	After	Before	After	Before	After
	per cent.	per. cent.	per cent.	per cent.	per cent.	per cent.
<6/60 ...	15	0	27	0	10	5.2
6/60-6/24	28	2.4	38	2.2	43	5.2
6/18-6/9	57	9.6	35	20.0	47	12.0
6/6 ...	40	88.0	0	77.8	0	77.6
No. of cases	47	Treated by atropine drops followed by occlusion	45	Treated by occlusion only	58	Treated by occlusion only

FIG. 9.

Again fusion training is delayed until vision is nearly equal in both eyes, but Swan and Laughlin (1944) have recently advocated it, when the vision in one eye is still much below standard. They state that provided that the target is large enough to correspond with the degree of vision, or to avoid the central scotoma—peripheral fusion can be established and appears to assist in the development of central vision.

Cases of heterophoria excluding large degrees of height, seem to improve adequately on duction exercises. I have found that quite a number, *e.g.*, 42 out of 78 cases, show a small degree of suppression—which is frequently alternating and appears only in the fixing eye. This suppression is no obstacle to their cure—because of the strong fusion sense which is always present.

False associated fixation presents one of the most difficult problems of training. In a group of 74 cases I found only one cure. The data showed that they all had, or regained, equal vision in both eyes—and showed a macular dominance, but with very little power of fusion. The subjective angle in every case except one

was under 10° —i.e., it corresponded to the retinal area between the disc and the macula, while in one case only the angle was 19° —i.e., immediately to the nasal side of the disc.

It has been suggested that in these cases operation should precede orthoptic training, but what is needed appears to be some form of training to develop peripheral fusion and by the use of large images, rather than to attempt to change macular vision from "false" to "true," a process which takes an exceedingly long time and has such strikingly poor results. Events might prove that the resumption of peripheral fusion would lessen the disparity in central fixation.

Finally, although the standard set by the British Orthoptic Council for a cure is quite commendable, I think it is far too high for every day purposes.

Costenbader (1945) in an analysis of the causes of failure in the treatment of squint, has suggested 4 grades of cure :—

- (1) British Standard (1939). Straight eyes, normal vergences—good fusion and stereopsis.
- (2) Good fusion and stereopsis, but varying from fusion to diplopia with Worth's test, and inconstant ability to bar read.
- (3) Fair fusion—fair vision and a small degree of residual strabismus.
- (4) Cosmetic cure—no fusion—a small degree of residual strabismus.

You will notice that fusion with convergence is only achieved in the highest degree of binocular vision. Stutterheim (1940) has advocated a new form of training based solely on convergence exercises. He starts, as it were, at the other end of the scale—and states that amblyopia may persist for a considerable time *after* the squint has been cured. Although he pours scorn on the recognised methods of orthoptic training, his book is worth reading, by way of contrast.

I have not attempted to analyse the surgical treatment of binocular abnormalities, but I think you will agree that good surgical results depend more on the moment chosen for operation, rather than on the exact adjustment of the angle of squint. I must end my discourse with an apology for the inadequate analysis of the subject of binocular vision, which I have been able to make. Perhaps some Middlemore lecturer of the future will be able to give us more advanced information. The present jig-saw of facts will then be transformed into a complete picture.

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THE REGENERATION OF WOUNDS OF EXTERNAL MEMBRANE OF THE EYE IN THE LIGHT OF NEW PATHOLOGICO-ANATOMICAL RESULTS*†

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PART I

Introduction

WHILE paying due consideration to the questions raised by ophthalmic surgery during the last war, we have to point out that insufficient attention was paid to the conditions at the moment of

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the wounding, and the further fate of the eye very often depends on them. In the Russian ophthalmological literature during the last war a criticism of the principles of the management of perforating eye-wounds was started (Levkoieva, Braunstein, Jenkin-Harkavy). From that point of view it is important to study (pathologico-anatomically), the eyes enucleated after trauma. We have studied during many years a large number of such eyes (about 6,000 altogether) enucleated as a result of different kinds of trauma; the study of war wounds during the "Patriotic War"—all these researches lead us to the conclusion that at present the first surgical measures do not guarantee a good healing of the wound. The pathologico-anatomical analysis shows that the wounds are left in an unsatisfactory condition, and this by itself, quite independently of other causes, may lead to fatal complications in the wounded eye.

The fact that those eyes had to be enucleated does not diminish the value of our material in forming those or other principal questions; first of all because the necessity of enucleation does not prove the extraordinary degree of the trauma itself. Because the eye is so small, forming a compact whole, relatively isolated, it is possible to study in it the causes and consequences of the changes better than in any other pathologico-anatomical object. This study brings us to the conclusion that parallel to the severe trauma, that certainly destroys many eyes, in a very important percentage of the enucleated eyes the *fons et origo mali* was not a severe trauma, but certain complications were the leading factors in the disastrous result. This is why the consequence becomes the cause, and this is why it is important to study and to fight it.

The fact of the enucleation does not exclude the fault of the ophthalmic surgeon, as far as the unsatisfactory state of the eye-wound is concerned; he struggles a long time before he decides on enucleation, even in large scale trauma, while the microscopical examination shows that his efforts were powerless and frustrated in cases, where from the beginning the right measures were not taken to close the wound. What result can show later the symptomatic and reabsorbing therapy, if under the well-formed scar of the conjunctiva the edges of the scleral wound are turned inwards or lie one on top of the other or the wound is still not closed? These conditions are the cause of constant irritation, reactive inflammation of the anterior part of the uvea and a source of haemorrhage in the wounded eye.

Following this reasoning and considering the importance of the correct approach by an ophthalmic surgeon to a fresh wound of the eye we analysed in our work the surgical principles up to date.

In the first instance we examined the question of infection in the eye injury. There are no serious bacteriological researches of this

point and we had to study it on the large pathologico-anatomical material at our disposal. We thought the solution of this problem to be so urgent, because at present the question of infection is a basic one in all surgical measures for the wounded eye. And after analysing the present methods of healing of eye wounds from this point of view, we came to the conclusion that the ophthalmic surgeon approaches this problem exactly like a general surgeon does to any kind of wound, and we started to search for a basis of rational therapy for eye wounds.

Finally we have examined a *sui generis* regenerative process of the external membranes of the wounded eye of considerable theoretical and tremendous practical importance. This factor at present is not appreciated and even not recognised by the clinician in his therapeutic or surgical measures. This is why we are going to characterise the essence and importance of those changes during the healing up of eye wounds. At the same time we have touched on the sore points of traumatic iridocyclitis—a general term that covers in clinical practice a whole range of changes in the wounded eye.

Accordingly the present paper comprises the following sections:—

- (1) Clinical and anatomical approach to perforating wounds of the eye.
- (2) A review of the methods of closing eye wounds.
- (3) The regenerative process of the external membranes of the eye.
- (4) Classification of eye injuries.

(1) Clinical and anatomical approach to perforating wounds of the eye. The most important factor which decides the fate of the eye is the exogenous putrifying infection that penetrates the eye at the moment of trauma, and, secondly, sympathetic ophthalmia. With the trauma as a background these two items decide the fate of the eye. The fear of infection defines the surgical measures when treating a fresh eye wound and the fear of sympathetic ophthalmia haunts the clinician all the time while healing is going on.

The infection is the foremost in the primary treatment of the eye wound. Some surgeons prefer to leave unsutured large wounds of the cornea and sclera, fearing the infection that penetrated the eye at the moment of trauma, and the same consideration of infection decides the method of operation when dealing with a fresh wound. The majority prefer to cover the wound of the anterior eye segment with a flap of conjunctiva (Kuhnt's method), and lately this operation completely replaced the suturing of the eye membranes

themselves with a close fixation of the edges of the wound—a method proposed more than 100 years ago and, according to the literature, beneficial in a whole range of major wounds.

In the further progress of the traumatic process the fear of sympathetic ophthalmia is so much in the centre of the surgeon's attention that it exceeds all other possibilities impressing itself clinically (precipitates, ciliary pains, hypotony) and leading to the decision to enucleate. The last resolution the surgeon accepts after long deliberation but usually with the same diagnosis of traumatic iridocyclitis, i.e., he does not think of infection and does not diagnose "endophthalmitis," but he fears sympathetic ophthalmia and therefore enucleates the eye.

First of all let us state that the clinician has an exaggerated idea about traumatic infection—the kind of infection that is labelled "endophthalmitis"—this kind of infection is also covering the reactive inflammation of the alteration phenomena, toxic inflammation, due to the presence of the foreign body, endophthalmitis phakogenica.

As a result the clinically diagnosed infection and the diagnosis of endophthalmitis very frequently are disproved by microscopical examination, and even an abscess of the vitreous proves often the result not of an infection, but of the previously described causes, like a foreign body, or endophthalmitis phakogenica—both of them not only clinically but also morphologically are very similar with some variations that only experienced pathologico-anatomists can recognise.

These are the results of our investigation about frequency of infection in the pathologico-anatomical material.

In 1941, among 263 enucleated traumatised eyes examined in the pathologico-histological department of Helmholtz's Institute in 56 (21 per cent.) we found endophthalmitis without any attempt to differentiate between the causes of it, as mentioned previously. In 1942, among 36 enucleated traumatised eyes we tried to separate the infective endophthalmitis and found it in 24 eyes (65 per cent.); in 1943, for 279 eyes, in 7.3 per cent. In 412 eyes enucleated as a result of air raids on Moscow we saw two cases of infection—4.7 per cent. In war wounds the percentage of infection is still lower—among 124 enucleated eyes—in 3.2 per cent. Altogether, for 779 eyes (including 1941) the percentage of infective endophthalmitis, according to pathologico-anatomical examinations, was approximately 5.7 per cent.

Dr. Jenkin-Harkavy on a smaller material of 100 enucleated eyes saw 12 per cent. infected (*Vest Ophthal.*, Vol. XXIII).

Prof. Braunstein also thinks that the prevalent opinion about the frequency of infection in eye trauma is exaggerated.

The microscopical study of wounded eyes leads us to a different opinion—you must not approach the perforating eye wound with ordinary general surgical methods. The ophthalmic surgeon must realise that the eye is a complicated closed system with very superficial correlation of the internal parts, where a small concussion, minimal processes of exudation or haemorrhage could be the primary cause of destruction of the eye. With wounds of the external membranes (cornea and sclera), because they are the capsule of the eye and so influence its form, all this happens more readily. With more or less considerable wounds of this capsule, especially with wide separation of the edges of the wound, this alone on purely mechanical grounds causes a disturbance of correlation within this delicate viscus with all the following consequences. When you study such eyes enucleated after a trauma and see widely separated edges of the wound wrongly healed up, with badly adapted edges, you are able to understand the colossal impairment, changes, disturbances of blood-circulation—all of them frequently accompanying the regenerative process of the eye and quite independent of the results of the trauma itself.

The primary task of the surgeon is to restore immediately after the trauma the disturbed correlation of the tissues in the eyes of a more or less large wound (more than 5—7 mm.), if, of course, he has the slightest hope of preserving the function of the eye and if he does not decide on immediate enucleation—an indication really present in a few cases, with a complete destruction and prolapse of all interior membranes of the eye.

We have analysed from this point of view 120 cases of war wounds. In all cases, where the wounds were in an unsatisfactory state, in 100 per cent. we saw a disturbance of blood circulation and very often a direct connection of the haemorrhages with the hole of the wound. Fresh repeated haemorrhages, occurring very late after the injury, are the proof of constant irritation resulting from a non-closed or badly closed wound.

And so the state of the wound is a factor of primary importance for a favourable healing result. Whatsoever happens later about the infection showing itself, we cannot decide in the beginning. If there is a foreign body—the closing of the eye wound cannot be delayed. Obviously, if an electromagnet is available we will first extract the foreign body and only after that will we close the wound. But if the electromagnet is not available—we must first of all close the wound properly and the foreign body will be extracted at the first opportunity later on. With a widely used diascleral approach the surgical incision very often does not correspond to the wound, and if we do not close the traumatic wound immediately we may be too late, even with a successful extraction of the

foreign body. In cases of added infection we have no reason at all to prove that leaving the wound open gives more chances for recovery and because of that to neglect to close the wound immediately.

As a résumé, we have to point out that in more or less large perforating wounds of the eye we have to consider not only the question of infection, but the whole clinical-anatomical position—that dictates an immediate reconstruction of the disturbed correlation on the anatomy of the eye, following the trauma.

Conclusion.—The healing process and the fate of the perforated eye depends chiefly on the primary surgical treatment.

(2) Review of the surgical methods of closing eye wounds. In the last war the question of closing eye wounds became of first importance.

It seems that it is worth while to re-examine the approach of the ophthalmic surgeon to wounds of the eye and its different membranes in the view of better results in healing because many sides of this question are still not decided.

From the point of view of the previously described anatomical factor the first task of the military or civilian surgeon is the earliest and best closure of the wound of the external membranes. What are the best methods in different wounds? They are only two—suturing of the sclera and cornea and the plastic of the conjunctiva bulbi by the method of Kuhnt. But we have to mention here that there exists a school of surgeons, who not only do not operate even on large wounds, but even do not excise the prolapsed membranes—and those membranes for certain do not allow the wound to heal up, as witnessed by unsuccessful operations with their incarceration in the scar, usually ending with enucleation. The fact is that in the great percentage of unsuccessful operations the bad result is due exactly to the membranes, caught in the scar and impeding the good healing up of the wound. The reason for this school's idea is the fear of infection that penetrated the eye with the trauma (Strachoff and his school). The partisans of this school do not enucleate the eye for a long time, even with very large wounds, and apply therapeutic measures, hoping eventually that large wounds of the cornea and sclera can heal up by themselves without any surgical interference. The microscopical examination of such enucleated eyes shows that as a rule spontaneous healing does not occur, and the wounds are in a highly dangerous state even without talking about the prolapsed membranes.

Let us now review the methods of closing the eye wounds. The simplest and most successful method—that of Baroti, published more than 100 years ago—is to suture the sclera, the edges of which usually are widely separated, because the sclera has a peculiar

morphological structure (plenty of elastic fibres, and moreover the muscles attached to it usually contract). As a result of the sutures the edges of the scleral wound are joined together, and are fixed in a proper position, thus having the necessary conditions for healing up. Dieffenbach sutured the cornea in 1847. In Russia, Ginsbur was the first to apply sutures to the sclera. The old Russian ophthalmic surgeons (Odzinof, Strachoff, Auerbach, Blagovechensky) previously always applied sutures in the wounds of the external membranes, if the wounds were larger than 5 mm. or were yawning. Varshofsky was a great partisan of suturing, and described cases when the sight later on was 0.3—0.5, although in the beginning it was nil (amaurosis). But now the suturing of the wounds in Russia and, according to the literature abroad is done much more rarely. This method not only did not become a usual one, but its results are described and recommended as rarities. H. K. Müller, in 1945, in his book, "Ophthalmologische Operationellehre," advances the importance of surgical closure of perforating wounds and recommends suturing (but not through the whole thickness of the sclera or cornea).

The second school thinks that it is advisable to suture only the conjunctiva, leaving the wound of the sclera alone. The method of conjunctival plastic by Kuhnt has lately become very popular and is widely employed in all kinds and dimensions of wounds, but unfortunately without the special individualisation demanded by Kuhnt himself. With a conjunctival flap the surgeon likes to cover even large wounds of the cornea and sclera, hoping that in this way the edges of the wound will remain joined. He is usually wrong.

The best method of controlling the success of this or other methods is the microscopical examination.

In 1941—1942, on the material supplied by air raids on Moscow, we could study the state of the wounds in severe injuries in 42 enucleated eyes; in 13 of them Kuhnt's method was applied, and the rest were treated conservatively. Moreover, we analysed in detail a large amount of material in 1941 in 263 enucleated eyes, where 63 eyes were operated on by Kuhnt's method, and in the rest no surgical measures were applied.

Let us speak first of all of the conjunctiva when discussing Kuhnt's method. Its usual rôle is described as follows:—

It protects the eye against secondary infection, and it is a very good corneo-scleral plastic material. But both opinions are wrong, as discussed above; even the danger of primary infection is not great, and that of a secondary infection still less so. And the importance of the conjunctiva as a good plastic material for formation of scars on the cornea or sclera is contradicted by actual facts,

because, according to the present literature, the regeneration of these membranes is produced by their local elements, and the conjunctiva does not form a scar.

The part taken by the conjunctiva, according to our material, is as follows :—

In 10 per cent. it still covered the wound. But much more often one can see only its débris present and then from time to time, hardly joined with the wound and therefore having neither a prophylactic nor a joining action. And even in cases where it is present it only covers the wound; it does not penetrate deeper and does not take part in the regeneration. The regenerative process of the external membranes of the eye goes on independently; the newly formed tissue, often filling up the wound and penetrating into the anterior chamber, sometimes goes even under the conjunctiva, but *vice versa* one cannot see any elements of the conjunctiva in the scar. But after Kuhnt's operation in the majority of cases epithelium is present in the scar; it proliferates largely and penetrates inside the wound, covering parts of the edges of the wound and growing to a considerable depth.

And so we have to reckon when performing this operation with the participation of the epithelium in forming cystoid scars, fistulae and real cysts; one sees all those things very frequently, not even mentioning rarer possibilities, such as the growing of the epithelium into the anterior chamber and encapsulating it with all its important consequences.

In all cases of Kuhnt's operation the prolapsed membranes were excised. This first and important step gave good results according to microscopical examination. In the majority of cases the membranes were thoroughly excised and were absent from the wound. But unfortunately the same good opinion cannot be given for the healing up of the wound.

And here we have to differentiate between large yawning wounds and small ones that clinically healed up with a small scar. In the first case the widely separated edges of the wound show that the flap of the conjunctiva cannot keep them joined. Even clinically we notice that on the second or third day after the operation the conjunctiva is off the surface of the wound, or the edges of the wound are not covered with the conjunctiva. Microscopically we often see that the edges of the wound were not joined together and the space between is filled up by newly formed regenerative tissue, or is simply yawning, and the edges may be already covered by epithelium.

In all these cases all the complications were present that are usually caused by non-restitution of the anatomical relations in the wounded eye, that were described previously (displacement

of the interior membranes, wrong joining up, disturbance of the blood circulation). All this is due to the fact that the surgeon does not pay much attention to the state of the wound when operating by Kuhnt's method. And this is the basic defect of this operation. Parallel with the yawning of the wound one sees a bad adaptation of the edges of the wound even when they are near one another. We have to point out that these edges (scleral as well as corneal) are rarely traumatised even with large wounds, and if sometimes one sees their splitting infiltration, or necrosis, then even at a small distance from the edges of the wound the membranes themselves are in a good condition. But generally speaking the correct apposition of the edges of the wound is extremely rare. In the overwhelming majority they are superimposed one on the top of another, or are healing up at an angle or are inverted into the anterior chamber; sometimes only one edge, sometimes both, as the result of the pull by scar formation or by folding and so further inversion. But in small wounds, operated on by Kuhnt's method, the yawning of the wound was small, and one would think that the flap of the conjunctiva would fix them satisfactorily. And clinically the healing up in those cases was going on smoothly and finished with a scar that looked good on the surface, but in those eyes that were enucleated later on was noticed a peculiar irritation, hyperaemia of the iris, miosis (exudation was rare), ciliary pains and, what is very characteristic, still later a flattening or even a subsidence of the surface, where the scar is situated—usually labelled as *atrophia bulbi incipiens* and the above described symptoms as the early stage of sympathetic ophthalmia. And with the diagnosis of traumatic iridocyclitis these eyes are enucleated. On microscopical examination one sees the previously described phenomena produced by the conjunctiva, epithelium and edges of the wound—all of them—less traumatised than in other cases and insignificantly changed; as far as the adaptation is concerned—in small wounds with a displacement of the edges—their adaptation is rarely a correct one. Together with the described changes, we saw in similar cases some interesting phenomena usually seen with Kuhnt's operation. We see a superficial joining of the edges of the wound and a good healing up of the superficial layers of the cornea with a hardly noticeable scar, nearly by first intention. By clinical examination from the external surface the defect is completely covered by epithelium and the scar looks perfect. But the healing up involves only the superficial layers and through the substantia propria, as a rule, the edges of the wound are not joined. And so the edges of a small wound, joined at the surface, gradually separate deeper—the deeper, the more so—and so in the picture of regeneration one sees always the figure of a triangle, with a base

towards the anterior chamber. This characteristic triangle, better or worse according to the section, was seen by us in more than 90 per cent. of all examined cases of Kuhnt's operation in small perforating wounds.

What is the meaning of this geometrical design? Its sides, equal in size, are representing the edges of the cornea; they limit the triangular defect very quickly, and even in the first 4—5 days the defect is fully filled up by new-formed tissue, growing from the edges of the wound. In the superficial layers, where the yawning is small, this tissue is insignificant, but the deeper it becomes the larger, according to the separated edges of the wound, forming whole layers on the internal surface of the cornea. In cases where there are defects of the endothelium of the membrane described, and also on the surface of the iris, it encapsulates the anterior chamber, penetrates deeper in the posterior chamber and sometimes even deep into the cavity of the eye, on the way soldering to itself all objects that it meets—iris, ciliary body, remnants of the lens.

Later on these bands become shorter and produce detachments of other membranes and a flattening of the surface of the sclera.

The anterior part of the uvea usually remains passive in this independent and powerful process of the super-regeneration, and the iris and ciliary body are only taken in by this rapidly growing tissue, but the signs of inflammation or exudation are absent. That is why we cannot reckon the clinical signs to be an iridocyclitis, and we will in the future have to reserve our opinion in this respect, taking into consideration the described morphological changes, that produce a compression, folding up and irritation of the eye, due to these powerful pulls on the inside of the sclera, that are seen clinically on the surface of the globe.

Coming back to the analysis of the methods used for covering the wound I have to examine the cases where no surgical measures at all were applied and the wound was left untreated. The result was even more unfortunate than with Kuhnt's operation, where at least an attempt was made to join the edges of the wound by a conjunctival flap. The yawning of the wound is larger; the edges of the wound are folded; and all the described complications are much worse. This is why the wound ought to be stitched up even in cases of very severe trauma. From the point of view of the anatomical approach the failure to join together or fix by one or other method the edges of the wound—even in large ones—means negligence in taking all the necessary steps to save the eye. Do we succeed in all that when operating according to Kuhnt? Following the well-known technique of this operation with all its modifications we have to conclude that the efforts of the surgeon are directed against the infection, but not to the fortification of the edges of the wound.

It is evident that if the above described triangular defect in the wound could be abolished and properly sutured by strong sutures, the regeneration of the wound would proceed within normal limits. It always happens when the edges of the wound are correctly joined. But in the cases where the regeneration process became ultra-powerful, in spite of the absence of infection or of danger of sympathetic ophthalmia, in spite of the relative insignificance of the wound itself, the eye was lost only because in our surgical



FIG. 1

The wound after Kuhnt's operation. The edges of the wound are displaced, one of them is covered from the surface by the debris of the conjunctiva. Both edges are covered with epithelium, on one of them is proliferation of the epithelium along its surface, this edge is inverted.

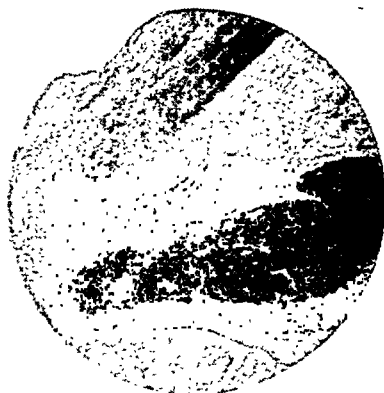


FIG. 2.

After Kuhnt's operation. The edges of the sclera are wrongly joined, are superimposed and taken in by the scar tissue.

measures in such cases, particularly when Kuhnt's method was employed, we neglected the necessary approach and so allowed a very frequent complication to arise.

This is why especially under battle conditions we have to resist the temptation of applying Kuhnt's method and/or to cease to use it completely, or to use it only in exceptional cases. The popularity of this operation at present gives to the surgeon the sensation of

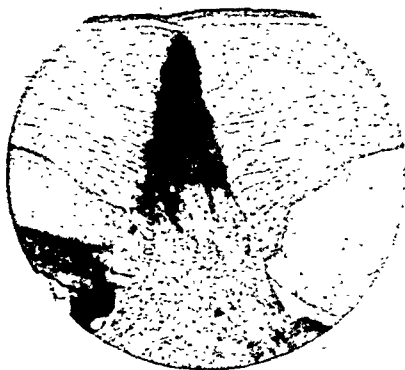


FIG. 3.

The triangle after Kuhnt's operation. The surface of the wound is closed, in the depth the edges of the wound gradually separate, forming a triangular defect that is filled up by scar tissue growing from the edges of the wound. The scar penetrates into the anterior chamber, forms a wide powerful band, growing deep into the cavity of the eye. On one side, the iris is taken in by the scar tissue.



FIG. 4.

A similar picture from another operation of Kuhnt.

safety and so limits his activity in the individualisation of his approach that was recommended by Kuhnt himself. As a result of our analysis, the basic requirement of our surgical measures ought to be a solid joining of the edges of the wound on the surface and in the depth right through. And the most advisable would be scleral and corneal sutures always with a prior excision of the all prolapsed membranes. After removing all elements that may be in the way of the healing up of the wound we have



FIG. 5.

No surgical measures were taken. The edges of the wound are widely separated. The iris is prolapsed and is joined to one edge of the wound. The other edge is covered with epithelium.

to join the edges of the wound so as to have a complete adaptation and to try to fix them in this position by strong sutures.

Must these sutures be dia-scleral or deep scleral right through the whole thickness of the sclera, or cornea, more or less deep? This question must still be studied by experiments on animals because even the technique of such sutures is little known at present, judging by the literature of the subject.

Such is the present position of this method. These views are not only reckoned to be obligatory—I speak only about the large wounds—but judging by the literature are not widely applied in Russia or abroad. Müller, in his previously mentioned work, recommended the applying of sutures in 1945 and said that whoever will apply this method will be surprised by the number of favourable results in comparison with cases where the wound is left to the natural process of scar formation. The fact that till now we have still to persuade surgeons to use this method was the reason of the writer of this paper reading no fewer than ten papers at different scientific societies during the war years.

And during the war years the Russian surgeons became more critical about Kuhnt's operation (Prof. Strachoff was always against it) and some advocated application of sutures (Katznelson, Kopp, Longinoff, Chechik-Kunina, etc.). In the Institute of Helmholtz there is a great change of opinion in favour of stitching and frequently the results are brilliant. In Dr. Chechick-Kunina women's department all wounds of the cornea and sclera are always stitched up and comparing with last year, when the majority of the cases were operated by Kuhnt's method, the frequency of enucleations fell by 8.9 per cent.

The ophthalmic surgeons in the near future will undoubtedly work out a rational surgical method of healing up eye wounds helped by the pathologico-anatomist. The duty of the latter is to show the unsatisfactory state of the matter, to give it a new basis and the right direction.

PART II

THE important question—when discussing perforating wounds of the eye—is the process of regeneration of these wounds. As was indicated in my previous paper there are peculiarities in the process of the healing of wounds of the eye, that are of enormous theoretical and practical interest for an ophthalmic surgeon, but the importance of those details is usually not realised when treating a wounded eye. And because of that the process of regeneration, which is necessary and important in every wounded eye, becomes not a bene-, but a malevolent factor. When the sclera and especially the cornea are perforated, the regeneration of them goes on in its own peculiar way, dissimilar from the usual granulation of connective tissues. First of all the blood vessels do not take any part in it; they are of course absent in a normal cornea, and in cases of keratitis of different origins, including the traumatic ones, the vessels penetrate into the cornea from the peripheral net. But the walls of the vessels remain passive in the process of regeneration and the latter can go on with a complete absence of blood vessels. This is contrary to the usual process of the connective tissue, where the regeneration usually goes per fibrovascular way. However, we do not see here the usual morphological picture of the usual cycle-development of granular tissue—the young immature cellular elements of the connective tissue with their later differentiation are absent. We saw, in fact, on wounds in man and in experiment on animals, where the wound, even 2 to 3 days after the trauma, is already filled up by narrow and long fibroblastic cells.

According to Wolfram and Boenig, Hañke and experiments of

Zazybiñ and Plitas, also of our own, it may be reckoned as proved, that the regeneration of the cornea is due to the proliferation of the corneal cells. The epithelium, to which the older authors starting from Donders ascribed the principal rôle, is of no importance in this process—it plays quite a different part. Nor is the episclera of much importance, or suprachoroidea or conjunctiva, contrary to opinions of other authors, including the writer herself—in her work on pathological anatomical changes after Gonin's operation (*Arch. f. Ophthal.*, 1935).

It is clear for us now that they take no active part in the cicatrisation and are not a plastic material for the regeneration of the sclera or cornea.

Our own researches in the regeneration of the cornea are partly connected with the morphology of the new tissue, but chiefly with the dynamics of the regeneration; we first saw it in the experiment with the transplantation of the cornea in the anterior chamber of rabbits (report read during the fourth session of the Central Ophthalmic Institute in Moscow).

I perforated the cornea of the rabbit and introduced at the same time in the anterior chamber pieces of the cornea of rather large dimensions; because the implanted piece was so large, I had to use some force to introduce it in the anterior chamber and so the edges of the wound were badly traumatised. As a result the implanted piece already on the fifth day—the earliest date of enucleation—was completely surrounded with new formed tissue, starting from the corneal scar, the scar itself by this time being already properly formed.

In this experiment we watched chiefly the implanted object; but the above described tissue was in the way and with the view of the isolation of the graft we started to introduce it through the pupil into the posterior chamber, but the quickly growing scar-tissue penetrated there too, surrounded it, and also any other organs in its way—Iris and the lens, for instance—and penetrated deeply inside the eye, in a horizontal direction, up to the ora serrata, and further, and so we could not isolate ourselves from this tempestuous growing tissue. This first made me study this extraordinary energy of growth of the corneal scar and also some other peculiarities—and these were the basis of our further studies of the regeneration of the sclera and cornea in cases of trauma of the eye.

The above described experiment with the transplantation of the cornea entirely corresponds to the wounds of the eye, our graft being similar to the foreign body, together with a considerable traumatisation of the edges of the wound.

When analysing the further stages of the regeneration, by studying eyes enucleated because of wounds of the cornea, we see how

the scar tissue, growing towards the deep inside of the eye, penetrates everywhere, where it does not meet any obstacles—first of all into the anterior chamber, where the foreign body itself already prepared the way. Here this scar-tissue at once becomes much larger, first because it is joined by new tissue—if Descemet's membrane and endothelium are also wounded—and secondly, because its own cells proliferate so quickly; this later process can be especially clearly seen on the periphery where the scar-tissue is in contact with the aqueous. Becoming very powerful, sometimes having the form of a band, the new formed tissue penetrates the posterior chamber, first going round and later enclosing the lens, and goes still deeper; and if the lens is also wounded, it provides a further stimulus for this new formed tissue. In some cases, this tissue follows the route of the wound right through the eye up to its posterior part. It has such a characteristic form, because the cells are basophilic in the initial stage, and especially because of their fibre-like structure, that with some experience you can recognise it even with a weak magnification. It is entirely different from the products of organisation and inflammation of the wounded eye, which are joined frequently in some degree to this basic process.

Later on the number of the cells in this tissue diminishes, they are less basophile; the tissue looks more pink, like the mature corneal tissue, sometimes structured even in real layers like a normal cornea. This final metamorphosis is the witness of organogenesis—very interesting from a theoretical point of view.

At present we will discuss chiefly the phenomena of practical interest to an ophthalmic surgeon; such is the presence of the regenerative tissue in the anterior chamber and the resulting increase of the intra-ocular pressure in a wounded eye. It is necessary to add that this produces one of the worst forms of secondary glaucoma. It is caused by the fact that the scar-tissue, after penetrating the anterior chamber produces whole layers of new tissue not only on the posterior surface of the cornea, but also on the anterior surface of the iris and so closes the angle of the anterior chamber and encapsulates the whole chamber altogether. Compared with other factors that usually close the angle of the anterior chamber, like the ordinary synechia, hyphaema, exudation—even the changes in the configuration of the iris, where it is possible for some holes still to be left free—the above described process is extremely dangerous, because with the encapsulation of the anterior chamber by this scar-tissue the angle is closed completely and it is obvious that in those cases all our therapeutic measures are useless, because this kind of tissue is not capable of being absorbed—like some of the other indicated pathological phenomena.

It is worth while mentioning here that a similar encapsulation

of the anterior chamber occurs by the ingrowing into it of the epithelium penetrating by proliferation from the wound. In those cases the anterior chamber is covered by a delicate veil of one layer of epithelium that can be seen only under a microscope. You cannot see it through the slit-lamp, while the more solid regeneration-tissue in band-form connected with the scar can be seen easily sometimes even with ordinary focal illumination, and it is this tissue that is sometimes erroneously diagnosed clinically, as exudation, pus, secluding tissue, etc.

I will speak only shortly about regeneration of the wounds of the sclera, because the principal processes are the same as in the cornea. The same stormy hyperproduction of regeneration tissue can be seen. It shows itself in the tendency of the scar to go deep into the eye by powerful bands or layers, going very far away from the perforating wound of the sclera. En route, this tissue, as in the anterior part of the eye, goes round all objects that it meets, and the latter also take some part in the process. You can make very interesting observations on different membranes of the eye—their morphological and potential peculiarities; their interaction with the basic process. But it is only a detail in the overwhelming process of fibromatosis. The bands, arising straight from the scar, could be so big as to go right across the eye, sometimes filling up completely the interior space, deforming, separating and growing into other membranes. The infiltration at this stage may be quite insignificant, if the eye is not infected and if the process does not go on towards sympathetic inflammation.

We have to point out that we see a similar process on the sclera after diascleral removal of foreign bodies, when the whole success of this operation is annulled by the described complications. The degree of the trauma to the sclera is the deciding factor. We do not think that the epithelium plays such an important part as was ascribed to it years ago.

But still there is a difference in the regeneration of the sclera and of the cornea. In the former we do not see what I have described, the struggle with the gradual alienation of the deeper layers, that is often seen in the cornea, especially after Kuhnt's operation. The wound of the sclera forms a defect of equal dimensions right through the membrane—a difference that undoubtedly depends on the anatomical peculiarities of those two membranes, especially the presence of Descemet's membrane, whose elasticity produces the gradual compression of the deeper layer—a process not seen in the sclera, where a similar membrane does not exist. In all surgical measures this difference must be remembered and this is why deep corneal sutures are even more necessary than deep scleral.

What do we see clinically in the typical cases of this regeneration? Frequently these are small perforating wounds, limited to the anterior part of the eye, with a well healed-up small external scar, with a stubborn continuous irritation that instils in the mind of the surgeon the fear of sympathetic ophthalmia and usually such eyes are enucleated with the diagnosis of traumatic iridocyclitis.

But the microscopical examination in this kind of trauma usually does not confirm these fears in the majority of cases: the inflammatory-exudative, and especially infiltrative processes in the iris and ciliary body are usually insignificant; the sympathetic process is seen only in 2-3 per cent.; intermediate forms are normal (10 to 12 per cent.); the primary importance belongs to the described super-regeneration process, whose particulars are so similar that they are becoming an artefact, being different only in some details, but not in essence. The iris and ciliary body usually remain passive; the reaction in them is insignificant; the iris and ciliary body are only joined to the super-regeneration tissue round them with consequent atrophy of their elements, and the clinical diagnosis of traumatic iridocyclitis in those cases proves to be a wrong one. And this general all-embracing clinical diagnosis is not exhausted by the pathological process of the anterior part of the eye. *And so, in cases of the above described changes in the wounded eye, we see an absolutely separate independent process, not noticed yet up till today, but with a peculiar tendency of its own.*

Proliferation of powerful bands of tissue inside the eye leads to atrophy of the bulb. These bands are the immediate continuation of the scar and are the products of super-regeneration of the wound.

How often do we see this process destroy the eye without evidence of other pathological changes? According to the statistics of our pathological histological department of war wounds, and also that of civilian wounds for 1943 (415 enucleated eyes) the above described traumatic process was seen in 23 per cent. (in civilian category in 29 per cent.). For battle wounds where the trauma is more heavy, where an increasing percentage perishes from the traumatic moment itself, the percentage is a little lower—16 per cent.

This shows that we are describing a factor of major importance, especially if we consider the higher percentage of blindness in perforating wounds of the eye, shown by the latest statistics (Preobrajenskaja, G.N.).

The grave danger to the eye from perforating wounds makes us pay special attention to the causes of this danger and to the measures to fight them.

This super-regeneration process, the principal one in more than 20 per cent. of enucleations, is an important object of study that will attract our attention in the near future.

Can we indicate now any points for the clinician? How to differentiate between this process and the other one, with the real danger of sympathetic ophthalmia? We think that we can indicate already one symptom, when with a long-standing irritation, we see on the surface of the bulb flattening, falling-in, sometimes even a pull from inside of this or any other part of the sclera or cornea, that coincides with the localisation of the scar. In those cases the sclera loses its peculiar globe-like form, sometimes in limited spots, sometimes on the large surface of the sclera; especially characteristic is this picture on the limbus, where the peripheral part of the cornea seems to be cut off, being drawn inside.

These phenomena, verified microscopically, show without any doubt that in this particular globe are present powerful agents, capable of deforming the bulb in the locality of the scar, consequently the process is due to super-regeneration and—as a result of a dense fibrosis with a consequent scar formation—deformation of the surface of the eye takes place. In this case the whole symptom complex, suspicious as to sympathetic ophthalmia, may be referred to the above described fibromatosis, that has nothing in common with the sympathetic one, and where the irritation and painfulness are probably due to compression and scar formation and therefore natural irritation of the nervous apparatus of the eye; this is, of course, merely a proposition, and we will have to verify it on the morphological and physiological analysis of the nervous tissue of such eyes. But clinically there is a good deal of similarity between these two processes, which are really quite different in their substance and we hope that in the crystallisation of the diagnosis the clinician will add many new points.

We must understand the peculiarities of this process, its morphology and pathogenesis to be able to fight successfully. Special attention ought to be paid to the structure of the scar; its extraordinarily quick and powerful growth, especially after the anterior chamber is penetrated, where there is a liquid milieu; also when the edges of the wound are separated or form an entropion, *i.e.*, their wound surface is facing the anterior chamber. In those circumstances this process becomes very intensive. The same intensification is seen when this regenerating tissue comes in contact with the substance of the lens, when its capsule is wounded—I described that previously; it seems to give an extra stimulus to the growing tissue.

All these circumstances are bringing us to the conclusion that the regeneration of both sclera and cornea are proliferative processes, where similar conditions are necessary—a liquid milieu, a good nutritive substitute, are for the cultures of tissues *in vivo*, but of the types of such cultures *in vitro*.

Our researches on the changes in the eye lead to the consideration of the eye as of a highly organised camera, where may be developed similar reactions. (This was recognised a long time ago, under the name of "autonomy of the eye"—translator's remark). The presence of good nutritive media, helped by the fibrin of the haemorrhage and its solution, products of eye-tissues damaged by the trauma, and, besides, the constant temperature and closed cavities—all these are conditions favourable for creating an "exclusive" chamber, but in addition with constant metabolism; in this connection we see in the eye the inflammatory processes and at the same time the purely proliferative ones, peculiar to the eye, as to a closed cavity containing liquid milieux of different chemical composition.

For a long time now histology has been busy with the growth and preservation of neoplasms and other tissue-elements—in the anterior chamber and the eye of animals. Unfortunately ophthalmology up till now did not appreciate the meaning of those experiments for better understanding of the pathological processes in the wounded eye, rather peculiar to this organ, as discussed above.

In 1941 in my paper on "spontaneous cultures of the tissue inside the eye" (Vestn. Ophthalm. Vol. XVIII, A. 5), I pointed out that—parallel to inflammatory processes and their reactions—in the eye take place also proliferative processes, peculiar to the eye, as a viscus where are present liquid milieux; a similar phenomenon was described by me in the enucleated eyes in different diseases, but seen quite by chance. We see most frequently in trauma of the eye (but also in other diseases), that the tissue elements show a real cytotypic growth, so characteristic in the tissue cultures *in vitro*. For instance, I described the presence in the anterior chamber of a culture of Schwann's cells, developed from the iris, probably from the sphincter, where are present ectodermal elements; this culture covered like a delicate veil the whole anterior chamber. Cultures of pigment-epithelium are seen frequently, proceeding into the cavity of the eye from the retinal pigment, or into the anterior chamber from the pigment epithelium of the iris; cultures of ciliary epithelium, lenticular capsule, etc. In the cavity of the eye the elements of the detached retina frequently show signs of intensive proliferation, and in accordance with the nature of this process and consequent variation of the exudation, the proliferation affects different elements of the retina. We saw also a typical growth of fibroblasts from a scleral scar in cases of war wounds. And, lastly, displaced pieces of the membranes of the eye act as a real source of tissue cultures; far removed sometimes from their primary location and meeting favourable conditions in the liquid

milieu they produce epithelium and continue to exist for many years.

I discussed all these questions because I wanted to make clear the development of this stormy regeneration process in the eye, impossible to understand otherwise. The uniform structure, extraordinary quickness of growth, conditions favourable to direct this growth deep inside the eye—those are reasons to reckon it as a culture of tissue, although some characteristics of the cytotypic growth are absent. But on the periphery of the quickly growing, very tight, brush-like band of the fibrous type tissue, in the places where it grows more free and comes in contact with a liquid milieu, one can see a different structure and separate figures of typical star-like fibroblasts, very similar to those we see in the scleral scar. Narrow, thin, arrow-like cellular elements, separating themselves from the common band and entering the liquid milieu look like typical elements of a growing tissue-culture.

When staining these spots for the intercellular tissue by the method of Snesev it is seen that we have to deal with fine argentophil fibrils, with the collagenous ones beginning to appear, and their disposition has an irregular mixed up character—and all of these not characteristic of a fibro-vascular type of regenerative connective tissue, but peculiar to the conduct of the inter-cellular substance in the tissue-cultures. This was also the opinion of Prof. Snesev, in whose laboratory my preparations were stained, and for whose help I am profoundly thankful.

What are the practical deductions of this reorientation of the surplus growing regenerative tissues of the external membranes of the eye towards tissue-cultures? We have, to use the experience of the scientists, who are working with the method of tissue-cultures, because they have worked out a whole host of conditions, favourable to the growth of tissue elements and *vice versa*. From our point of view we are advising not to allow the presence in the wound of the liquid milieu, of the nutritious medium, like lenticular masses—and especially we have to fight—when healing up the wound—against the penetration of the regenerative tissue into the anterior chamber, *i.e.*, a proper closing of the wound is the first condition of a good result. Besides that we have to try to regulate and to depress the regenerative process by chemical and other methods, not only different from those that are usually employed for the stimulation of wounds, but taking into consideration the peculiarities of the above described process.

In the first paper we have made already corresponding practical suggestions about the necessity of active surgical methods when treating a wounded eye: as soon as possible we have to reconstruct the damaged co-ordination and to sew together the

edges of the wound. The analysis of the regeneration-process points to the importance of detailisation of this method, because it is not sufficient to put stitches on the sclera or cornea—it ought to be done in such a way that the edges of the wound are close together, with the correct adaptation, and right through the whole depth of the wound.

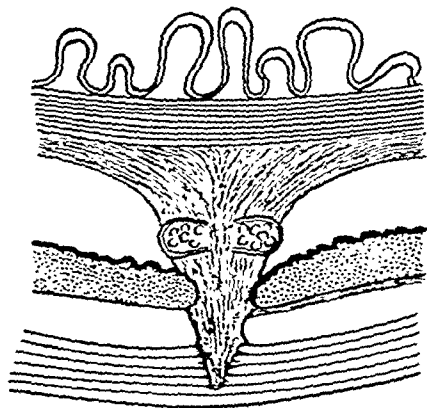


FIG. 6.

A scheme of corneal regeneration—starting from the triangular scar, the band grows gradually thicker and penetrates deep into the vitreous. On its way it takes in the iris, goes right through the traumatised lens and forms a powerful layer structurally similar to the corneal substance. It joins and detaches the retina.

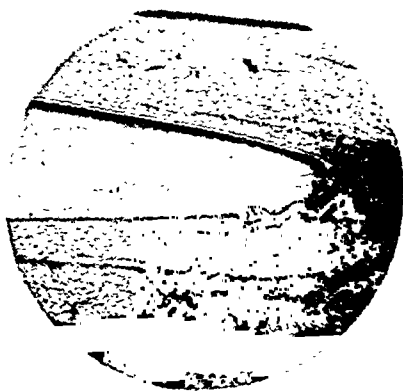


FIG. 7.

Encapsulation of the anterior chamber by a band of new formed tissue (on the right), that goes round the angle of the chamber and forms layers on the anterior surface of the iris.



FIG. 8.

A contracted scar of the cornea after Kuhnt's operation with proliferation of the epithelium from the surface. The edges of the wound are wrongly adapted; the left one is joined to the right one only in the upper part; further down the edges are separated, forming a triangle. A large band of newly formed tissue—a continuation of the scar grows into the interior. The more recent immature part of it can be seen with strong magnification in the following photo.

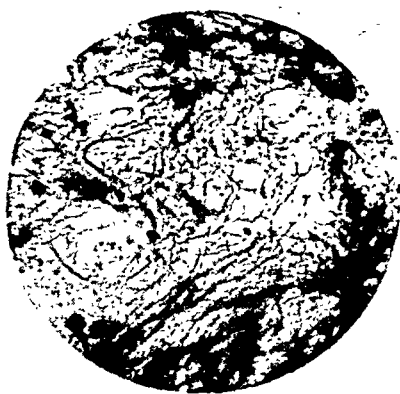


FIG. 9.

The inter-cellular substance of Fig. 8 stained by Snezarev's method. A mixed net of fine argentophyl fibrillae; here and there are passing single thicker collagen fibres.

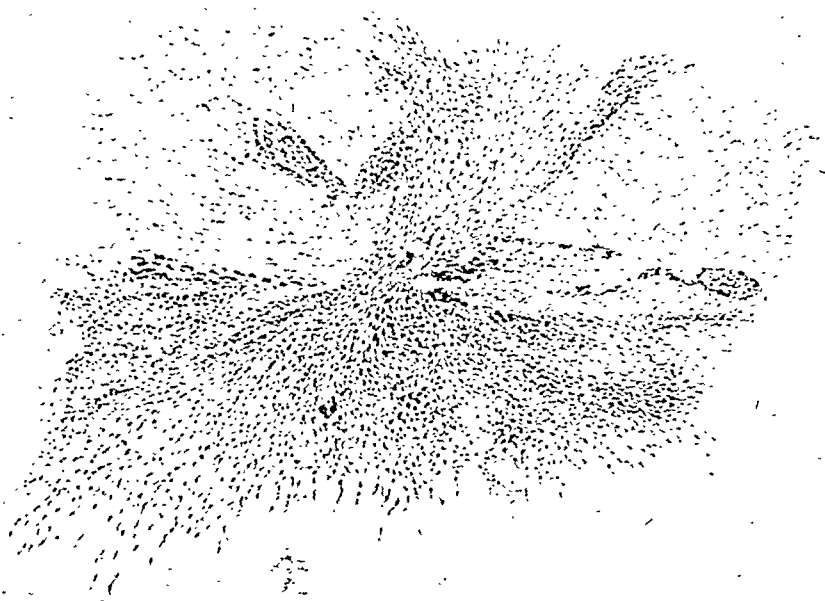


FIG. 10.

Scleral scar in a war wound. In its deep parts, in the liquid milieu of the perichoroidal space (the retina and choroid are detached)—a typical culture of fibroblasts.

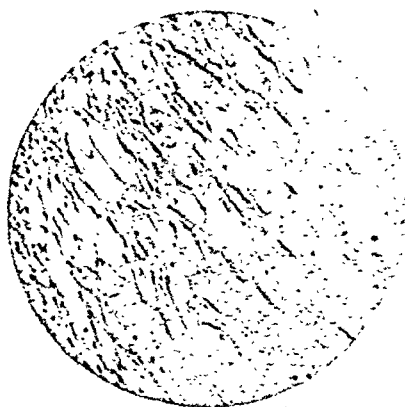


FIG. 11.

The same cells with greater magnification.

Unfortunately I cannot mention in the present paper a host of clinical instances and illustrations—they would form a whole atlas—where an incomplete closure of the wound and irregular adaptation of the edges with their entropion in the anterior chamber, and especially their transmutation, are the source of powerful layers and bands inside the cavity of the eye.

But, even with all that in mind, we have to study a whole range of separate moments in the healing of the wound before we shall be able finally to adopt a method of stitching.



FIG. 12.

In the wounded eye, from the scar of the iris, a fine veil of tissue, attached to the interior (posterior) surface of the cornea, grows into the anterior chamber.

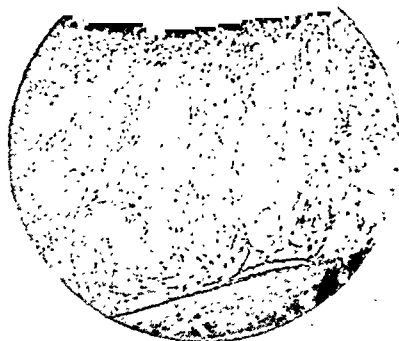


FIG. 13.

The same veil with greater magnification.

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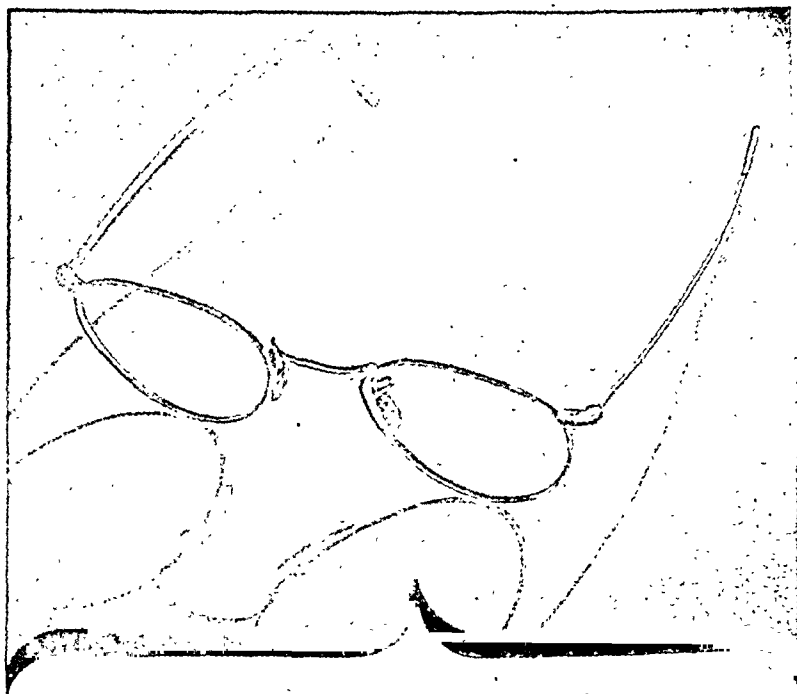


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FIG. 14.

Cellular elements of the same cultures.

These are my conclusions :—

(1) The regeneration of the sclera and cornea is not the ordinary granulation process of connective tissue, that is going by the fibro-vascular way, with its peculiar cycle of development of the cellular elements, but a proliferation *sui generis* of similar cellular forms that morphologically are near to tissue cultures.

(2) The dynamics of this process are characterised by an extraordinary intensity, as a result of which—in unfavourable conditions—is seen a surplus-growth, chiefly as a result of the penetration of the regenerative tissue in the liquid milieux of the eye.

(3) Following the described peculiarities of this process and the idea that the eye, as a closed chamber, is ideally adapted for tissue-cultures *in vitro*—we ought to use the experience of histologists and their ability to regulate the growth of the tissue-elements.

(4) We ought to undertake the clinical and experimental research of the best methods of closure of eye-wounds, and also of the regulation of the regenerative process of the healing of the wound; besides surgical perfection, we ought to treat it as analogous to the tissue-cultures.

(5) In connection with surplus regeneration of the eye-wound is seen an autonomous fibromatosis in the eye, pathologically quite a different form of disease, that has nothing in common with sympathetic ophthalmia, but clinically very similar to it, and with our present knowledge, still labelled under the common diagnosis of traumatic iridocyclitis, which ought to be differentiated from it in the future.

PANOPHTHALMITIS IN A PREMATURE INFANT TREATED BY STREPTOMYCIN*

BY

L. B. SOMERVILLE-LARGE

DUBLIN

THE FOLLOWING is an account of panophthalmitis in a premature infant caused by *bacillus proteus* and treated with streptomycin.

The patient was a male premature child weighing 4 lbs. 5 ozs. at birth. He was born in the Rotunda Hospital, and I was called to see him there in the Premature Ward when he was eleven days old. The ocular history was that inflammatory swelling in the lids of his left eye had commenced twenty-four hours previously and was rapidly increasing. He had had a superficial skin infection of his lower abdomen not reaching the umbilicus. This had cleared up rapidly on 30,000 units of penicillin injected over a twenty-four hour period, and was at that time completely cured. The umbilical cord had not fallen off. This separation occurs normally on the fifth or sixth day, but in this case was not complete until the twelfth. The baby was progressing satisfactorily, taking feeds well and putting on weight.

On examination of the condition I found an inflammatory oedema involving the lids of the left eye. This was well marked on the upper lid, but was only slightly apparent on the lower. The pre-auricular gland was not enlarged. The eye was proptosed. No conjunctival discharge was present. There was intense ciliary injection. The cornea was hazy. The anterior chamber was half full of hypopyon which lay over the pupil, no pupillary margin being visible. There was no red reflex. The condition was diagnosed as panophthalmitis with a possible infection of the orbit. Panophthalmitis occurring after birth is usually regarded as due to an infection of the umbilical cord. Its occurrence at this period of life is rare. No case has occurred in the Rotunda Hospital during the last twelve years (approximately 55,000 births). The right eye was normal.

Mr. R. R. Woods, the Ear, Nose and Throat Specialist to the hospital, kindly examined the case, but no source of possible nasal infection was found.

General examination revealed that the umbilicus was infected and also that there were two subcutaneous "abscesses" in the skin of the lower abdomen. These latter did not wholly resemble abscesses as they were not accompanied by signs of inflammation

* Received for publication, March 29, 1947.

but appeared rather as small herniations through the abdominal wall. They were, however, aspirated for examination. A swab was taken from the umbilicus. Both examinations revealed *bacillus proteus* in pure culture. It was considered that the primary infection had taken place through the umbilicus.

Immediate operative treatment consisted in passing a fine Graefe knife into the orbit, and a search being made with a probe for pus. No pus was found. The proptosis remained unchanged. Three days later a needle was inserted into the anterior chamber and pus removed for bacterial examination. Here again *bacillus proteus* was grown in pure culture.

Immediate therapeutic treatment consisted in the administration of sulphadiazine by mouth and penicillin by injection. A total of fifteen grammes of the former and 170,000 units of the latter were given within the first six days, when they were discontinued and streptomycin started. The *bacillus proteus* proved bacteriologically to be resistant to penicillin.

During the first six days under the above therapy the ocular condition became steadily worse. Severe oedema developed in both



FIG. 1.



FIG. 2.

lids. The conjunctival vessels became very congested and pannus commenced above. This pannus eventually covered the upper third of the cornea, and superficial blood vessels also encroached on the rest of the corneal periphery. The centre of the cornea developed a deep opacity. The child's general condition, however, remained highly satisfactory. There was no rise of temperature and weight was gained steadily.

Bacillus proteus is resistant to penicillin and the sulpha drugs but is highly sensitive to streptomycin. There was some delay in getting this drug, and no information as to dosage in a premature child could be obtained. Treatment was carried out as follows: One gramme of streptomycin was dissolved in 10 c.c. of sterile water and 1 c.c. was injected intramuscularly every three hours. This was continued for twenty-one hours, when the dose was reduced to 0.5 c.c., which was given three-hourly for sixty-six hours. Towards the end of this period the baby was bottle feeding so slowly that tube feeding had to be started. There was also a loss of weight (from 4 lbs. 14 ozs. to 4 lbs. 12 ozs.) and a rise in temperature (99.8 to 100°) for two days. It was uncertain whether

these symptoms were due to streptomycin, but some reduction was thought advisable. The dosage was therefore reduced to 0.25 c.c. every three hours, and this was continued until a total dosage of two and a half grammes had been given. The treatment with streptomycin covered a period of six and a half days.

Streptomycin was commenced at 3 p.m. one afternoon, and on the following morning for the first time there was a marked improvement in the clinical appearance. The lid oedema had greatly subsided and the eye was open. On the second day the skin folds had returned, and on the third all inflammation had gone from the lids leaving only some slight oedema. The condition of the eye itself remained unchanged. The subcutaneous abscesses disappeared soon after the streptomycin was commenced and required no local treatment.

On the ninth day from the commencement of streptomycin the globe perforated at about six mm. from the limbus between the inferior and lateral recti. Evisceration was carried out on the same day. The vitreous was found to be grossly infected. Swabs from the site of perforation, from the anterior chamber, and from the vitreous were taken. All were found to be sterile on culture.

The pathological report on the cornea and uveal tissue removed is as follows:—

The piece of cornea sectioned shows a considerable amount of proliferation of the epithelium. At one point this epithelium and Bowman's membrane have been broken through by a haemorrhage coming from a new vessel lying just under Bowman's membrane. A considerable number of new vessels can be seen at different levels, the majority lying anteriorly. Scattered chronic inflammatory cells are present mostly in the anterior third. At one point there is an aggregation of these cells involving the neighbourhood of Descemet's membrane and the posterior fourth of the adjacent substantia propria. In another area the whole corneal thickness is diffusely infiltrated with chronic inflammatory cells. The piece of uveal tissue consists mainly of choroid—a small part of the ciliary body is also present. The entire tissue is infiltrated uniformly, but not heavily, with chronic inflammatory cells, round, plasma and epithelioid. Some polymorphs are also present suggesting that the active inflammatory phase had not completely passed. Normal choroidal structure is unrecognisable. Proliferated pigment is present, passing through the section. Blood vessels are very numerous, the greater number containing erythrocytes. Some scattered haemorrhages are also present.

By the finding of bacillus proteus in the anterior chamber (in pure culture) prior to commencement of streptomycin treatment, and by the negative cultures obtained from the highly infected

globe after treatment, it may be inferred that this drug is capable of permeating the uveal tissue.

It is of importance that infection of the umbilical cord by a non-virulent organism is capable of producing panophthalmitis, and this without any general symptoms of septicaemia. Meningitis can, however, occur in infants from bacillus proteus, and this was a complication that was feared in this case.

The child is now three and a half months old, has remained in perfect health and is gaining weight steadily. No ill effects from the streptomycin treatment are apparent.

Summary

A case of panophthalmitis in a premature child following umbilical cord infection by bacillus proteus is described. An account is given of the treatment with streptomycin which is considered to have overcome the inflammation in the uveal tissue.

I am grateful to Dr. Ninian Falkiner, Master of The Rotunda, and Dr. R. Collis, Paediatrician to the hospital, for their advice and permission to publish this case. To Dr. Hinkston's invaluable assistance, and to Sister Moran who looked after the case I am greatly indebted. I am much obliged to Miss Thompson for her fine photographs, and to Dr. McCrea who kindly carried out the pathological examination.

ON MENINGEAL REACTIONS IN SYMPATHETIC OPHTHALMITIS*

BY

L. CORCELLE

AGEN, FRANCE

IN 1934† I showed for the first time that meningeal reactions occurred in the course of sympathetic ophthalmitis, of which the interest is such as to warrant fresh emphasis. Before my paper appeared we had recognised the extra-ocular complications of sympathetic ophthalmitis, but their frequency was not very great and their description so very changeable that they were considered to be of secondary importance by most authors. However, Coppez, in his article on sympathetic ophthalmitis in the last *Traité d'ophtalmologie français*, gave them a few lines and cast doubts on

* Received for publication, January, 1947.

† Contribution à l'étude de l'ophtalmie sympathique. L. Corcelle, Thèse, Bordeaux, 1939.

their nature. However, deafness, studied by Garrigou, Snellen, Rogman, de Wecker, Peters, Patry, Blatschek, Komoto, Truc, Serege, Sgrosso, and Calogero, was well understood. The importance of this complication, it is true, is variable and has escaped those authors who only studied the pathology of the disease. It should be remembered because one must suppose that the cause—allergy or infection—is susceptible of overrunning the framework of ocular pathology. On the other hand several very troublesome neurological complications have been observed.

Risley, Galezowski, Deutschmann and Mooren have reported the occurrence of convulsions. Delirium has been noted (Deutschmann, Terson, Sérége).

Colin and Duthil have published a case of severe sympathetic ophthalmitis with a spinal syndrome, pains and fibrillary contractions. The word meningitis has rarely been used. Renard, alone, has suggested it.

Snellen and Sérége have published observations where they use the term meningitis, but they did not examine the cerebro-spinal fluid of their patients.

Also my own observations were the first to allow of the affirmation of a meningeal reaction in sympathetic ophthalmitis. They appear to have received confirmation since their publication and it is actually possible to draw up a balance sheet of the new knowledge they represent and the importance of which can no longer be neglected.

Here is an account of three particularly typical cases to establish the good foundation of their existence.

Case 1. A Spanish workman, aged 63 years, sustained an accident at his work on January 28, 1933. A block of wood struck him in the region of the right orbit. He immediately complained of great pain and that he could not see with the damaged eye.

Examination showed: R.E. large hyphaema, dilated immobile pupil, lens luxated into the anterior chamber, no wound of the eye. T. —1.0. V. = P.L. L. eye, normal.

General examination disclosed renal insufficiency. Blood examination, normal. Negative serological tests for syphilis.

February 1, 1933. The lens was extracted by Snellen's scoop: slight loss of vitreous. The after-course was not satisfactory. The operation wound did not heal well; a prolapse of iris persisted in the upper part of the wound, pericorneal injection was very intense.

February 2. Hypopyon. The blind inflamed eye was enucleated on March 4, 1933, that is 33 days after the accident and 31 after the lens extraction.

Three days later the patient complained of diminution of sight in the left eye. Vision was now 1/20. Grey exudate showed in the pupil and seclusion was practically total. Tension was normal.

At the same time the patient complained of a feeling of lassitude and some shivering fits. He had no rise of temperature. The sympathetic iritis progressed in spite of all treatment.

March 22. An iridectomy was performed but the gap was soon closed by exudation. The tension was subnormal. The patient became deaf and complained of headache. Neurological examination was entirely negative and in particular he showed no sign of meningitis. Lumbar puncture was performed on April 1, *i.e.*, 24 hours after the onset of sympathetic uveitis, and gave the following results.

Cytology, 17,512 (lymphocytes).

Benzoin colloidal, normal. Albumen, 0.30: glucose, 0.75: chlorides, 7.40.

Twelve days later a fresh examination showed:

Cytology, 4,654 elements. Albumen, 0.30: glucose, 0.78: chlorides, 7.30.

On April 28, the vision was totally abolished, the eye was atrophied and the patient attempted to commit suicide.

Microscopical examination of the enucleated eye showed nodular leucocytic infiltration in the operation wound forming the "inoculation chancre" described by Redslob.

Case II. A man, aged 28 years, was wounded in the left eye on November 4, 1938, by a splinter of metal which perforated the front part of the eye.

On examination, there was a perforating scleral wound at "3 o'clock." In the wound was a small uveal prolapse and a bead of vitreous. Large hyphaema present. L.V.=0. Right eye, normal, V, 10/10.

On admission the uveal prolapse was cut off and the wound covered with a conjunctival flap. The after-course was normal, yet the eye remained blind. On January 4, 1939, it was decided to remove it, as signs of sympathetic irritation were apparent.

On January 15 the patient complained of limitation of sight in the right eye. Vision was found to be 8/10. Very intense pericorneal injection was present with precipitation on the posterior corneal surface, with posterior synechiae.

January 30. All signs increased in intensity. Vision, 1/10. Neurological examination negative. Lumbar puncture gave the following results:

Cytology, 63,3 lymphocytes: albumen, 0.50: glucose, 0.63: chlorides, 7.50.

Visual acuity improved to 10/10 on April 11. The patient considered himself cured and went back to work.

September 7, 1934. He presented signs of ocular hypertonus. T. +50. This yielded to medical treatment. Lumbar puncture gave normal figures.

Microscopical examination of the enucleated eye showed the anatomical lesions of "chancre of inoculation."

Case III. A man of 67 years of age, a healthy looking farm labourer was examined for the first time on June 27, 1946. He said that at the age of ten years he had sustained a perforating wound of the left eye. No surgical treatment had been undertaken and the globe retained merely poor perception of light. Four months before his visit to me he had received a fresh contusion of the left eye, but no perforation. Yet the globe became inflamed and painful and irido-cyclitis lasted a month. When "cured" perception of light was entirely absent.

After 3 months visual troubles began to affect the fellow eye. There was slight photophobia with vision reduced to 1/20.

Examination showed: Left eye, divergent, shrunken stump. The cornea showed deep greyish infiltration, with a number of new vessels.

The pupil was closed with old yellow exudate. There was complete seclusion with iris atrophy.

Right eye. Pericorneal inflammation. Many small precipitates on Descemet's membrane seen with the aid of the slit-lamp. Grey exudate rimmed the pupil and tied it down to the anterior lens capsule. The fundus was normal.

General examination showed nothing in particular. The examination of the nervous system was wholly negative. Lumbar puncture performed June 29, 1946, *i.e.*, 7 days after the onset of illness of the right eye, gave the following results:

"Cellule de Nageotte" 16, el 6 mm. 3, lymphocytes. Albumen, 0.60: glucose, 1.60: chlorides, 7.02. Khan and Meinicke reactions, negative.

June 29, 1946. Left eye enucleated. During the illness there was no fever.

July 7, 1946. Eight days after enucleation a second lumbar puncture was done with the following results:

"Cellule de Nageotte" 190, el mm. 3 (lymphocytes). Albumen, 0.80: glucose, 0.90: chlorides, 7.02. Blood sedimentation, 1st hour, 21 mm. Second hour, 43 mm. "Les Vernes resorcine: 17."

Between June 6 and August 8, 1946, the patient received 20 intravenous injections of sodium salicylate, of bevitine and 10 injections of staprolysat. The visual acuity was unaltered at the end of the treatment.

A third lumbar puncture was made on the 30th day of the disease and showed Cellule de Nageotte 32, el mm. 3. Albumen, 0.45: glucose, 0.45: chlorides 7.15.

At the present time the pupillary exudates are absorbed. The patient declares himself satisfied that the sight may be a little improved.

In the last three months I have seen two other cases of sympathetic ophthalmitis, the notes of which are as yet incomplete but they present meningeal reactions of the same kind. These observations have been confirmed by several authors.

In 1936 Espildora Luque found in 5 cases of sympathetic ophthalmitis a meningeal reaction traceable only through lumbar puncture.

In the first case there were 120 cells, in the second, 92, in the third, 5, in the fourth, 4, and in the last 20, but this author has not stated at what particular time the lumbar punctures were performed.

In 1945 Beauvieux and Bessi re published a note of a marked meningeal reaction with 543 lymphocytes.

In 1946 Toulant reported to the Academy of Medicine four cases with meningeal reactions.

Legroux published a case with meningeal reaction of 8.8 lymphocytes on the 96th day of the disease.

Clinical Description

It is now possible to sketch the clinical picture of meningeal reactions in sympathetic ophthalmitis.

At the start there are no neurological signs. Headaches of frontal type have been stressed in some cases. (Beauvieux and Bessi re, Corcelle).

Espildora Luque has described them and mentions the possibility of vomiting. He thinks that headaches are the rule in the lymphocytic meningitis of sympathetic ophthalmitis.

Redslob has emphasised their importance, but has not established a relation with the meningeal reaction. When they exist they are of frontal type and very severe.

In Beauvieux and Bessi re's case they were occipital with exacerbation at night. They sometimes precede the onset of inflammation in the sympathising eye.

Deafness is frequently noticed. It does not appear always to be linked to the existence of a meningeal reaction. In six cases I have observed it thrice.

Vestibular depression, which I have previously noticed, is to be found by systematic examination but it is inconstant.

One has also to mention the existence of a rise of temperature ($38^{\circ}\text{C}.$) of short duration. The first and third of my cases showed a brief rise of temperature with general lassitude. These symptoms are inconstant and are not sufficient to warrant the existence of a meningeal reaction. Therefore the onset of lymphocytic meningitis is indicated by lumbar puncture. This has been done in fixing the

date of the appearance of clinical signs in the sympathising eye at the end of 24 days (Corcelle, 1); 15 days (Corcelle, 2); 7 days (Corcelle, 3); 10 days (Beauvieux and Bessière); 96 days (Legroux).

One can affirm after these first results that the meningeal reaction may perhaps be regarded as contemporaneous with the appearance of clinical signs in the sympathising eye. In my 3rd case it was 166 elements on the 7th day, but by the 15th it had risen to 190 elements. By the 30th day it was 32 elements at which time the clinical signs were greatly improved. This evolution is not always calculated on that of the anatomico-ocular lesions. In Beauvieux and Bessière's case it was massive (543 elements) but yet remained at 400 elements on the 74th day of the disease, when the affection seemed healed. The total number of lymphocytes in the C.S.F. appears proportional to the intensity of the affection. Grave cases are accompanied by a marked reaction but one cannot base a prognosis on it. However, in my first case I noted 174 elements and the patient became blind. In case 3 the meningeal reaction was 190 and the patient was cured. I have since seen a case in which the meningeal reaction was 88 elements which ended in blindness. Also in the case of Espildora Luque the numbers reached were few (56 or 21 elements) but we do not know enough of the clinical course in this case. Enucleation does not by any means influence the lymphocytic meningitis. In three of my published cases excision of the exciting eye was done when lumbar puncture allowed the study of the C.S.F. Conversely, excision could not be incriminated. In my third case the existence of lymphocytic meningitis had been proved before surgical intervention. Examination of the centrifugalised deposit is not the only interesting subject for study. Chemical examination offers particulars which I wish to emphasize. The albumen is usually normal, it can, however, rise proportionally to the numbers of lymphocytes: 0.80 for 190 elements and later 0.60 166 elements. On the other hand hyperglycosis in the C.S.F. is clearer. It was raised in my 3rd case to 1 gr. 80. In the other cases it remained at a lower figure.

Serological reactions for syphilis were negative in my cases. Espildora Luque in his cases noted 3 positive reactions.

Two points are of importance to be clear about in ending this description of the meningeal reactions in sympathetic ophthalmitis. Are these reactions constant and are they specific for sympathetic ophthalmitis?

The small number of published observations comprising an examination of the C.S.F. does not allow me to answer the first question in the affirmative. I know of a case where normal C.S.F. is mentioned, but the account does not permit any indication of the day of the disease when the lumbar puncture was made. In all

the cases I have seen in ten years I have always found a meningeal reaction.

Espildora Luque has examined the C.S.F. of patients suffering from a penetrating wound of the eye without finding very important modifications of the fluid, except in one case where a reaction of 3 elements existed and also a case of iridocyclitis of the damaged eye.

What is the meaning of this meningeal lymphocytosis? One very interesting observation is that of Beauvieux and Bessière for it comprises the existence of a retinal detachment, thus approximating sympathetic ophthalmitis to the syndrome of Karada. Now we know that this latter syndrome is itself accompanied by meningeal reactions, so it is logical to make this approximation, already presented by other authors (Magitot).

In a recent report devoted to uveitis of unknown origin* we have proposed considering sympathetic ophthalmitis as a lymphocytic uveo-meningitis.

This would appear to be due to a filtering virus as yet of unknown origin. The pathological theories, bacillary or allergic should be re-examined in the light of this new semiology. We should not overlook so important a sign of other diseases due to a filterable virus such as herpes and zona which I call lymphocytic meningitis. In particular the bacillary theory appears untenable, the meningitis perfectly curable and silence being far from the usual attacks of the Koch bacillus.

The path of transmission of this agent of a probable virus nature is itself illuminated by a new light. One should consider the infection as general (Fuchs, Muller, Marchesant, Filsenthal), and striking by way of the blood stream the particularly receptive tissues: uvea, meninges, internal ear. The way of the chiasma appears little less probable from the fact that lymphocytosis is so nearly contemporaneous with the lesions of the sympathising eye, which my first observations had not allowed me to establish.

At the same time many points are still obscure in the study of the meningeal reactions of sympathetic ophthalmitis in default of a sufficient number of examinations of the C.S.F. of patients.

It is with the object of arousing interest in these new examinations that this article has been written.

* Les Uveites de cause Inconnue, Bessière et Corcelle. *Soc. d'Ophtal. du Sud Oest*, Dec. 1946.

CONJUNCTIVITIS DUE TO EXPOSURE TO
DIMETHYL-SULFATE*

BY

H. J. STERN

JERUSALEM

A WORKER in a pharmaceutical laboratory presented himself complaining of bloodshot eyes which had existed for six days. He hardly felt any discomfort apart from an occasional slight itching; no lacrimation or secretion were present.

On examination both eyes were found to be bloodshot, the bulbar conjunctiva being injected only in the area exposed to the air. Neither the palpebral conjunctiva nor the bulbar conjunctiva normally covered by it showed any changes. The line of demarcation between the hyperaemic and the normal bulbar conjunctiva was rather sharply defined. The cornea was normal. Apart from the hyperaemia, the slightly oedematous conjunctiva had a peculiar opaque aspect, the superficial vessels only being clearly visible. Especially on the slit-lamp this opaqueness of the bulbar conjunctiva was striking.

A conjunctival smear did not show any organisms. A drop of adrenalin solution 1:1000 reduced the congestion considerably and the conjunctiva took the aspect of a opaline glass.

The clinical picture—hyperaemia and infiltration of the exposed bulbar conjunctiva—together with the information as to the occupation of the patient, suggested a chemical agent as the cause for the condition. This substance would probably be present in the atmosphere in the form of a vapour or fume; dust would be washed into the inferior fornix and exert its irritating action there, while the fornix was entirely normal.

The patient had been engaged in work entailing the use of dimethyl-sulfate, a substance which is used to introduce the methyl group into certain chemical compounds.

Dimethyl-sulfate ($\text{CH}_3)_2\text{SO}_4$, is a colourless, heavy oil which has a boiling-point of ca. 188°C . Direct application to the skin produces a very intensive reaction, and its vapours are known to have an irritative effect on the mucous membranes. Prolonged exposure to it may cause serious nervous symptoms.

The noxious effect of dimethyl-sulfate on the eyes has been subject to a number of publications at the beginning of this century.

Weber¹ was the first to recognize its toxicity and published three cases: two were workers who had been exposed to dimethyl-sulfate fumes in the course of their work; one of them died from lung

* Received for publication, April 24, 1947.

complications. The third one was a chemist who had upset a bottle of the substance on his clothes and had suffered severe burns. All three cases showed a violent conjunctivitis, the first one with necrotic patches on the palpebral conjunctiva. No corneal involvement was noted. Weber experimented on some animals and found that dimethyl-sulfate rubbed into the skin caused local burns and a mild irritation of the eyes due to the fumes. Inhalation of the fumes caused watering of the eyes after 10 minutes; after $1\frac{1}{2}$ hours the conjunctiva was violently inflamed and the corneal tissue had become opaque. Ingestion of the substance did not cause any eye symptoms.

Erdmann² published the case of a chemist who displayed a violent conjunctivitis after having been exposed to dimethyl-sulfate fumes. While other workers had remained unaffected, he suffered from a marked reddening of the tarsal conjunctiva and a violent injection and oedema of the bulbar conjunctiva. The cornea showed some fine, greyish opacities in the area of the lid aperture. After 24 hours the cornea was opaque and the epithelium became detached in big vesicles. The condition receded slowly and the cornea became entirely clear, but with the corneal loupe some fine opacities remained visible.

The same author³ published some more cases all of which showed irritation of the tarsal and bulbar conjunctiva; some of them displayed also corneal complications.

Adams and Cridland⁴ published another case of a chemist who had started to complain of ocular symptoms some four hours after having been exposed to dimethyl-sulfate fumes and was still displaying irritable and watery eyes after three weeks when he was first seen. The ocular conjunctiva in the palpebral aperture was much injected and considerably oedematous. The palpebral conjunctiva was normal and the corneal epithelium sound, but there were some keratic precipitates present in both eyes. The patient suffered from a loss of taste and smell, and there was a doubtful contraction of the visual fields. The patient recovered entirely within three months. The authors assumed the presence of an affection of the cranial nerves because of the neurological signs:

The case published here resembles most the last one but seems to have been milder still as he presented neither the keratic precipitates nor the neurological complications of the previously quoted case. The patient was engaged in supervising a process in which dimethyl-sulfate is dropping slowly into a container in which the substance to be methylated is stirred electrically and develops a considerable temperature. The fumes of dimethyl-sulfate seem to have been responsible for the conjunctival irritation. The mildness of the symptoms and the fact that the patient did not connect his

eye condition with his occupation made the diagnosis difficult. Only the peculiar localization of the conjunctival irritation in the area of the lid aperture made it possible.

The patient was advised to stop working with dimethyl-sulfate, and the condition improved considerably. When he started after a few days to work with the substance the condition appeared again. Treatment with bland lotions and ointments failed to bring relief but bathing of the eyes with borax solution, which neutralizes the substance, improved the bloodshot appearance and itching of the eyes. Changing over to a different job brought about complete cure. It seems worth mentioning that, as in the case published by Adams and Cridland, other workers in the same laboratory who had previously done exactly the same job had never been affected.

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ANNOTATION

Patron Saints of the Eyes

An abridged reprint of the paper entitled as above, by Koch, is published in the Supplement to the Sight-Saving Review, 1944. The original was the author's thesis for membership of the American Ophthalmological Society and appeared in February last year. Here is given the lore of some of those saints who, in the middle ages, were canonized for miracles in connexion with sight. These saints are four in number: St. Lucy, St. Odille, St. Clair and St. Augustine of Hippo. St. Lucy was a virgin martyr of Syracuse, born c. 283 A.D. Her name was invoked principally by those suffering from external diseases of the eye. The legend of St. Odille should be familiar to those who know the Ingoldsby Legends. In this paper she is said to have been born to Adalric, Duke of Alsace and his wife. Odille was born blind c. 660 A.D. and narrowly escaped being put to death by her irate father. She is reputed to have recovered her sight during adolescence, became a nun and later the Abbess of a convent where she lived in the odour of sanctity for nearly a century. Both St. Lucy and St. Odille are commemorated on the same day, December 13th.

St. Clair, whose day is November 4, was a priest and martyr of the IXth century. He is invoked, for the most part, by those in Northern France, in cases of ophthalmia. There are other Saint

Clairs also, as well as an Italian St. Clare of Assisi, in this case, a lady.

St. Augustine was born A.D. 354. His day is on August 28 according to the Roman Martyrology and June 15 in the Russian church. He seems to have been a vicious youth both in morals and in religion; but he was converted and became Bishop of Hippo.

A well documented paper full of interest.

BOOK NOTICE

Sensory Mechanisms of the Retina. With an Appendix on Electroretinography. By RAGNAR GRANIT, M.D., Director of the Nobel Institute for Neurophysiology, Professor of Neurophysiology, The Royal Caroline Institute, Stockholm. Geoffrey Cumberlege, Oxford University Press. 1947.

An idea of the scope of this book will perhaps be best conveyed by a brief historical review (naming only protagonists) of the discoveries of the physiological processes giving rise to visual phenomena.

In 1876 Boll discovered visual purple, which was exhaustively investigated by Kühne from 1877-82. Its genesis and chemical properties were elucidated by Wald (1934-39) and Lythgoe (1937-8). In 1903 König first estimated its absorption curve and pointed out its agreement with the scotopic luminosity curve. Kühne had already proved that rhodopsin was contained only in the rods of the retina, and that visual impulses were initiated by photochemical stimuli.

In 1868 Schultze found that cones predominated in the retinae of diurnal, and rods in those of nocturnal animals; and in 1898 Parinaud attributed night blindness to deficiency in the rod mechanism. In 1904 von Kries brought forward a vast array of arguments in favour of the Duplicity Theory that the rod mechanism is responsible for vision at low intensities of light, and the cone mechanism for vision of higher intensities and for colour vision.

Du Bois Reymond's discovery of the "negative variation" in nerves (and also the resting potential of the eye) in 1849 may be regarded as the starting point of electrophysiology. Since then overwhelming evidence has accumulated that even if changes in electrical potential are not the actual cause of nerve impulses they are a quantitatively accurate reflection of the actual cause. Holmgren (1865-82) and Dewar and McKendrick (1873-7), discovered the electroretinogram (ERG); and Gotch (1903-4), with the capillary electrometer, obtained the first ERGs which embodied all the features now known to us. Valve amplification has provided instruments fast enough to follow the rapid action potentials in the nerve

from an end organ. The retina was the first sense organ to be analysed by it (Chaffee, Bovie and Hampson, 1923). By it Adrian in 1928 proved that every sense organ reacts to an adequate stimulus by discharging a series of brief action potentials of constant size through its nerve fibre.

The ERG is a composite curve which integrates the complex electrical responses to stimulation in the retina. In this respect it differs from the comparatively simple responses in the optic nerve to the same stimulus. The typical ERG (Fig. 1) shows four chief alterations in the curve—a short negative change (*a*); a rapid

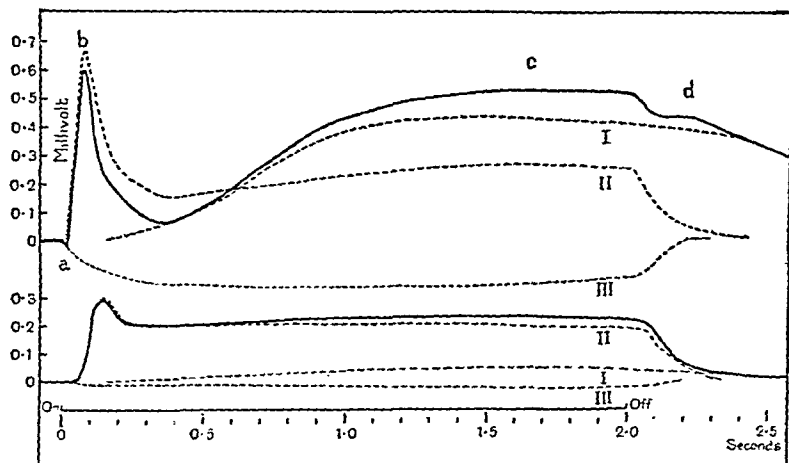


FIG. 1.

Analysis of the E—electroretinogram at two intensities—upper: 1.4 ml., lower: 0.14 ml.—dotted lines: PI, PII, and PIII.

positive wave (*b*); a slow and gradual positive wave following the fall of *b* (*c*); and an off effect, a positive change (*d*). The unexpected positive effect of the removal of the stimulus is itself evidence of the composite nature of the whole response. Granit has found two chief types of ERG: the E-retinogram in mammals (man, cat, dog, rabbit, guinea-pig, rat, mouse) (Fig. 1); and the I-retinogram in amphibia, fish, reptiles and birds (Fig. 2). The former seem to show a dominance of positive, excitatory, and the latter of negative, inhibitory components; but the responses in each are complicated by the preponderant type of retinal receptors (rods and cones), and the condition of adaptation.

The analysis of the ERGs shows that a minimum of three components suffices to explain a large number of the results obtained from various forms and conditions of stimulation; but it must be regarded as merely a first approximation. Einthoven and Jolly (1908) and Piper (1911) discussed three components, but Granit's

analysis (1933) is the most exhaustive and best established. His three component are PI, responsible for the *c* wave; PII for the *b* wave—both positive responses; and PIII for the *a* wave, negative and inhibitory.

Characteristics of the E-retina.—Ether successively eliminates PI, PII and PIII, anoxia removes PII, leaving PI and PIII; produces a negligible response at low and large at high intensities: the latent period of PIII is identical with that of the *a* wave. PI diminishes or disappears on light adaptation, and PII can be isolated by decreasing the illumination in the scotopic eye.

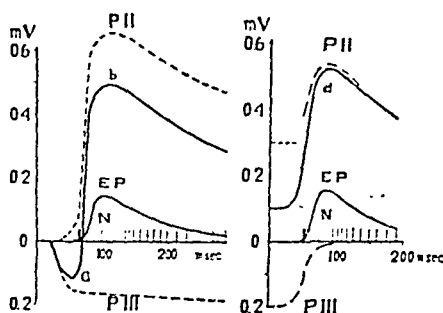


FIG. 2.

Analysis of the I—electroretinogram of the light adapted frog at "on" and "off"—dotted lines: PII and PIII (there is no PI in the light adapted frog retina); EP: electronic potential; N: nerve response; a, b, d: electroretinogram.

Characteristics of the I-retina.—PI is very slow and large in scotopia, absent in photopia: PIII and *d* wave larger in photopia. Adrenalin causes great increase in the latent period and duration of the *b* wave; it stimulates PI and increases the *d* wave. KCl in glucose eliminates PI and PII; the isolated PIII increases with increased intensity and prolonged exposure, and the optic nerve discharge is eliminated: the *a* wave is proved to be the commencement of PIII. Alcohol depresses PIII, so that it, after KCl, leaves a purely positive response. PII cannot be isolated, as in the E-retina. The large *d* wave of the photopic I-retina is due to diminution of PIII plus re-activation of PII. The initiation of both the *b* wave (PII) and the *d* appear to be due to pre-excitatory (*a* wave) and post-excitatory inhibition. The process responsible for PIII does not penetrate the optic nerve; hence the *a* wave is represented only by an inhibition of impulses, and PII is alone responsible for the electronic potential in the nerve.

PI is chiefly, if not exclusively, localised in the rod pathways, and may originate in the rods. Thus, the *c* wave is absent from pure cone eyes (turtle, tortoise, horned toad), and also in the frog with long wave-length stimulation. PIII is ontologically the oldest component (Keeler, Sutcliffe and Chaffee, 1928), and may originate in the neuroepithelium. It is most marked in cone eyes, but its potential reflects the Purkinje shift, so that it is also a feature of the rod system (Therman, 1938). Its greater development in cone retinae indicates the influence of amacrine cells. (The cones in the human retina are each supplied with a separate amacrine cell, Greeff, 1900). Its latent period diminishes with increased area of retina stimulated, suggesting that it is influenced by interaction in the

retinal synapses (Adrian and Matthews, 1925). "The whole picture of the behaviour of PIII during and after illumination suggests a piling up of inhibition during stimulation followed by its sudden destruction at "off" resulting in a release of excitation (Granit). "The complications of the retinal response at "on" and "off" are due to the intervention of pre- and post-excitatory inhibition, continuously active processes which are of extreme importance to the visual act. The discovery of the part played by inhibition in the retinal response, particularly with regard to the "off" effect, is the most important contribution that has been made by the electrophysiological method to the study of vision as a whole" (Granit).

PII is the most variable and complex response: "it represents the sum total of a number of processes with different latent periods and rates of appearance, and which produce different amounts of positive potential." Its reaction to chemical agents shows that it does not originate in the rods and cones. It probably represents the result of stimulating bipolar cells, as modified by their relationship to individual or grouped cells and cones, and their interaction with horizontal and amacrine cells. It is important to remember that some bipolar cells are connected to both rods and cones (Polyak, 1942). PII is greatly reduced by light adaptation, so that the rods are more efficient generators of it than the cones. Yet the size of the *b* wave accurately follows the scotopic and photopic luminosity curves in the dark- and light-adapted eyes of the frog, which therefore show the Purkinje shift (Granit and Wrede, 1937).

The E-retina is dominated by rods, so that the *b* wave is reduced in photopia. Yet PIII is much better developed in the cone than in the rod pathway: thus in man short wave lengths produce a typical E- retinogram (*b* wave with a small or no *a* wave) due to rods, whereas long wave lengths give a well marked *a* wave followed by a small *b* wave due to the cones (Adrian, 1945).

The I-retina is dominated by cones. In it PIII is dominant in pure cone eyes, and there is very little adaptation, though the pigeon ERG, dominated by cones, shows the Purkinje shift (Granit, 1942). In the mixed I- retina the scotopic curve shows small *a*, good *b*, appreciable *c*, and small *d* waves. PIII is characteristic of cone, PII of rod retinae. PIII is relatively greater than PII in all retinae in the photopic state. In other words the photopic eye is more inhibited than the scotopic.

"The dark adapted eye is highly sensitive and slow, like a ballistically recording galvanometer, integrating the total quantity of energy reaching it. After adaptation to light the retina is less sensitive, but very much faster and capable of differentiating between light and darkness much better than before" (Granit). In the dark adapted retina the rods must in some way be capable of damping cone activity, possibly by "occlusion" (Sherrington), the rods using

all available pathways and thereby excluding the cones. The rod system is a mechanism for integration, the cone system for differentiation (locus, changes in area and illumination intensity, discrimination). The nature of the synaptic organisation is probably more important than the character of the receptor cells.

Optic Nerve Responses. Hartline (1932) by microdissection on the retina and Granit (1939) by microelectrodes applied to the retina near the disc have succeeded in obtaining responses from single nerve fibres. These confirm the all-or-none law by giving discharges of equal size (unless the frequency at high intensity is such that each impulse falls within the refractory period of its predecessor). They also confirm the fact that increase of stimulus causes merely increased frequency of discharge. Prolonged stimulation leads to adaptation, with consequent diminution of frequency.

Hartline found three types of response in the frog: (a) "on" fibres—initial burst of impulses, maintained discharge, no "off" effect (20 per cent.); (b) "on-off" fibres—responding only at "on" and "off" (50 per cent.); (c) "off" fibres—responding only at "off" (30 per cent.). In the rod retina of the guinea pig Granit (1942, 1944) found 90 per cent. "on" fibres and no trace of inhibition. They have maximal sensitivity at $500\mu\mu$ and a scotopic luminosity curve, increasing in the short wave-lengths on light adaptation, possibly due to transient orange. The maximum frequency was 100 per second. The cat has only about 20 per cent. "on" elements, but 80 per cent. "on-off." The maximum frequency may reach 400 per sec. The rod retina shows least sign of synaptic modification. The rods give rise to long trains of impulses at a low frequency, and follow changes of intensity within narrow limits. The cones provide a mechanism of differentiation capable of responding to a wide range of intensity with variations of both frequency and impulse pattern. This response occurs both at the onset and at the cessation of illumination, so that the slightest change in the stimulus situation is translated into a new type of message to the brain.

Colour Vision. The microelectric technique has fully confirmed the identity of the scotopic sensitivity and the visual purple absorption curve, with its maximum at $500\mu\mu$, in a very diverse collection of animals (frogs, cats, rats, guinea pig). The photopic curve of retinas with enough cones corresponds with the photopic luminosity curve with its maximum at $560\mu\mu$. Granit calls these the scotopic and photopic *dominator* curves respectively.

In the light adapted eye some elements give sensitivity curves differing from the dominator curve in that they have different maxima and show humps on the curves. Exhaustive investigation has shown that these can be analysed into a finite number of curves—*modulator* curves—which together make up the dominator curve for

the animal. Such modulator curves are shown in Fig. 3. Fig. 4 shows the theoretical synthesis of the human photopic luminosity curve.

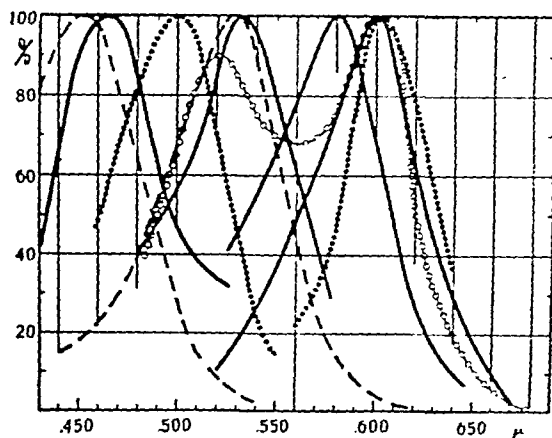


FIG. 3.

Modulator curves. Dots : rats; broken lines : guinea pig; continuous lines : frog; o-o : snake.

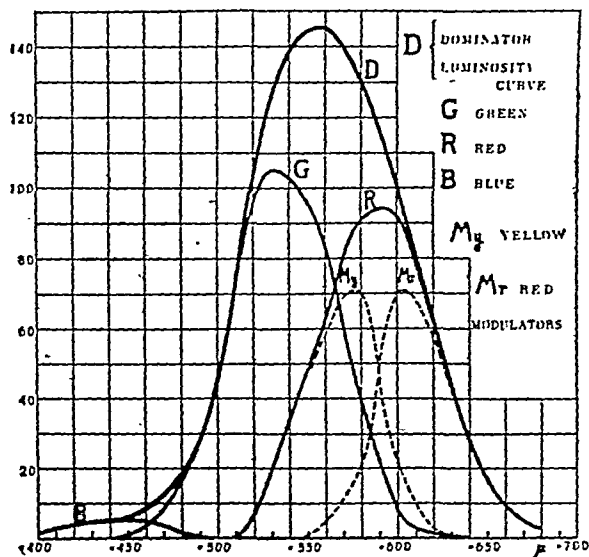


FIG. 4.

Synthesis of human photopic luminosity curve. D, luminosity curve; B, G, R, blue, green, and red fundamental sensation curves, the latter made up of two modulator curves, M_g and M_r .

The dominator—modulator theory shows that the scotopic and photopic curves are indeed true luminosity curves, *i.e.*, they represent

brightness differences. Both dominator reactions cover too large a part of the visible spectrum to give the higher centres any cue for colour discrimination. The modulator mechanisms are activated by light from specific central regions ($440-470\mu\mu$, $520-540\mu\mu$, and $580-600\mu\mu$), and thus give the higher centres their cues for colour sensations. In this respect they strongly support the trichromatic theory. On the other hand, the units of colour reception are much narrower modulator curves than the three fundamental sensation curves, which represent gross averages. Nature uses at least 6 or 7 colour mechanisms (2 red, 2 blue, and 2 or 3 green). The dominator—modulator theory affords explanations of facts which are not easily reconciled with Young's theory, *e.g.*, desaturation of the brightest parts of the spectrum, reduction of brightness on adaptation to coloured lights, summation of luminosities when two colours are mixed, the luminosity curve of deuteranopes, etc.

Hitherto our knowledge of visual phenomena has been due almost entirely to the psychological impressions derived from various forms and conditions of light stimuli. These gave little or no clue to the fundamental physiological processes involved or to their sites in the pathways from eye to brain. The first fundamental physiological fact was the discovery of visual purple and the photochemical nature of the effective stimuli. This led to the discovery of the identity of the scotopic luminosity curve with the absorption curve of visual purple and the rôle of the rods.

Granit's great contribution to the physiology of vision is the revelation of the processes in the retina and optic nerve underlying not only scotopic but also photopic vision. He has shown that the electroretinogram is the only measurable index of what takes place in the retina in response to changes in illumination. He has further elucidated the enormous importance of the synapses in the retina in both the scotopic and photopic pathways, which as elsewhere may be of a chemical nature. It must be a matter of satisfaction to him, as a disciple of Sherrington, to have been able to bring forward so much evidence of summation, facilitation, inhibition, occlusion, and so on in the retina, thus confirming physiologically its nature as a part of the central nervous system. His researches are of outstanding value not only to physiological optics but also to neurology in general.

As already mentioned his work necessitates modification of the trichromatic theory. The same applies to the duplicity theory. The "ideal" rod, to use his term, still survives as the scotopic receptor: but there are probably cone-like rods which are light resistant and are capable of mediating responses in the photopic state, probably by means of a modified visual purple.

It has always been difficult to understand how the relatively simple impulses of the optic nerve can convey to the brain the

qualitative differences of vision, and it has been customary to refer these to "higher centres." But, as Granit says, "whatever the sensation under consideration, whether flicker, colour, or brightness, the higher centres are no magicians capable of taking rabbits out of an empty hat." The complex fundamentals have been proved to have their origin in the retina. Differences of frequency and pattern provide qualitative differences in the optic nerve impulses: and it may be that, since there is a difference between the action potentials of fibres of different diameter (Gasser and Erlanger, 1924), the modulator paths may be made up of fibres of fixed size.

J.H.P.

CORRESPONDENCE

MYOPIA AND PSEUDO-MYOPIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I apologize for not having answered Mr. F. S. Lavery's letter in the February number of the *Brit. Jl. Ophthal.* before.

The boy he mentions had appealed and was re-examined, and was rejected a second time before I saw him. I fear that he has misunderstood me, for I did not wish to imply that it would be in the boy's interest that he should be accepted now. I agree that it is possible that he may later become myopic, but I merely quoted his case as the starting point of my interest in this condition.

Personally, I consider that it is wiser for such cases to wait, and if, when they have reached the suitable age, their vision, etc., is satisfactory, they should avail themselves of the "Public Schools" entry facilities.

Yours faithfully,

J. P. SPENCER WALKER.

3, SELBORNE ROAD,

HOVE 3, SUSSEX.

April 24, 1947.

NOTES

Contact Lens Society A scientific meeting of the Contact Lens Society was held on April 21, at 65, Brook Street, with Professor Ida Mann in the chair. About 160 members were present. A discussion on the training of contact lens practitioners was opened by Mr. Williamson-Noble, followed by Mr. K. Clifford Hall, F.S.M.C., Mr. Frank Dickinson, F.B.O.A. (Hons.), and Mr. D. W. A. Mitchell, F.B.O.A. (Hons.), F.S.M.C., D.Orth. Others who contributed to the discussion were Messrs. G. D. McKellen, Professor Ida Mann, C. L. G. Jenkin, Dr. Minton, A. E. Hogg, L. Crawford, R. A. Jones, and A. J. Forknall.

A vote of thanks to the speakers was proposed by Mr. L. Evershed Martin and seconded by Mr. C. L. G. Jenkin.

During the course of the evening three films were shown: Mr. Davis Keeler's which showed particularly the manufacturing side; the Feinbloom film which had just been flown from the United States and was shown in this country for the first time; and the London Refraction Hospital film.

* * * *

Ophthalmology in South Africa PROFESSOR H. WEVE, of Utrecht, is in Johannesburg at the time of writing. He will lecture before the Ophthalmological Society of South Africa and will also visit Cape Town and Durban.

Dr. J. Graham Scott, late of Glasgow, has started practice in Johannesburg and has been appointed Part-time Ophthalmologist to the Mines Benefit Society. Basil Graves has also been in South Africa and is now at George, Cape Province.

* * * *

Corrigendum DR. P. H. BEATTIE'S appointment, noted on p. 320, should have read Assistant Ophthalmic Surgeon.

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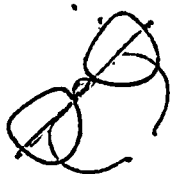
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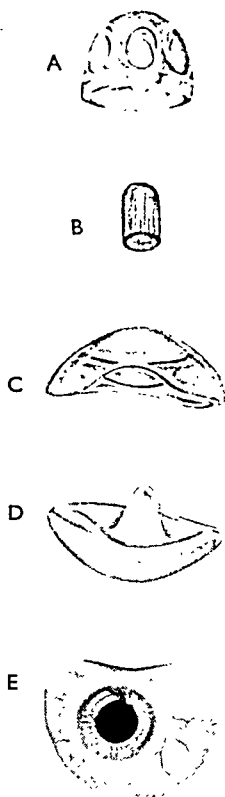
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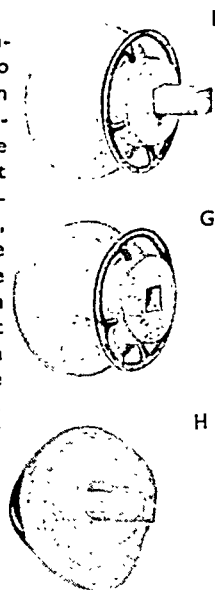
CUTLER'S TWO IMPLANTATION SETS

The illustrations depict the two new plastic implants devised by Dr. Norman, L. Cutler of Willmington, Delaware, for use after enucleation to impart maximum movement to the prosthesis. These on the left depict the original "Basket" implant and accessories, those on the right the later "Ball & Ring" implant, which provides a positive mechanical contact between the implant and the prosthesis.



A. Shews the "Basket" implant, B. the button used to maintain the depression during healing, C. the retainer-shell placed inside the lids to keep the implant centred and prevent prolapse of the conjunctiva, D. the retainer used after the removal of B., and E. the final prosthesis, which, in structure is similar to D., but made to fit the depression after the shrinkage of the tissue has taken place.

F. Illustrates the newer "Ball" implant shewing the ring to which the rectus muscles are attached. G. shews the implant with the pin removed for imbedding in the final prosthesis H.



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THE BRITISH JOURNAL OF OPHTHALMOLOGY

JULY, 1947

COMMUNICATIONS

FAMILIAL CATARACT WITH EXTENSIVE PEDIGREE CHART*†

BY

I. LLOYD JOHNSTONE

WORCESTER

THE purpose of this paper is to present an interesting and hitherto unreported genealogy of familial cataract, to describe the biomicroscopic features of the cataract and to discuss a few published papers in comparison with the case herein reported. The cataract is not present at birth but has been observed as early as six years and may be as late as thirty, forty or even fifty years of age in showing itself by causing serious loss of vision. Cases of early and late development co-exist so that it is impossible to say that the cataract shows the characteristic of anticipation. Its features are so characteristic that a member of the family possessing the cataract may be identified with certainty on examination of the eyes.

* A paper read before the Midland Ophthalmological Society at Dudley on April 6, 1945, with a few subsequent additions.

† Received for publication, February 4, 1947.

or feather-like branches radiating from the centre, thus somewhat like concussion cataract, and (d) flakes and dots in white, yellow, green or blue, throughout the cortex but always separated from the anterior capsule by a sub-capsular clear zone, the zone of disjunction, (e) accentuation of the shagreen of the anterior capsule. The earliest signs observed so far are visible only with the slit-lamp.

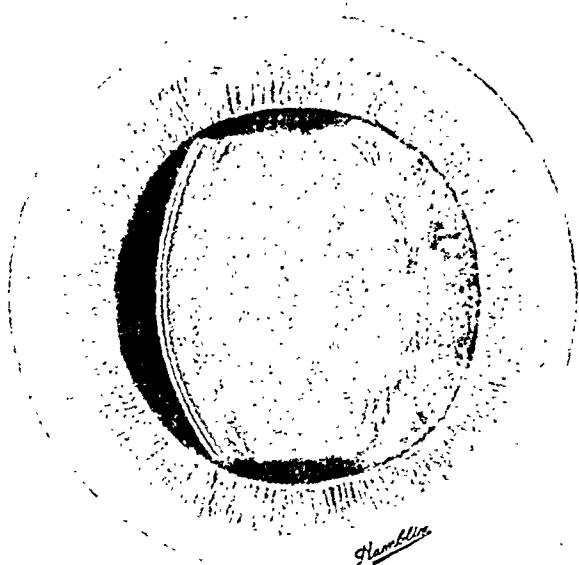


FIG. 4.

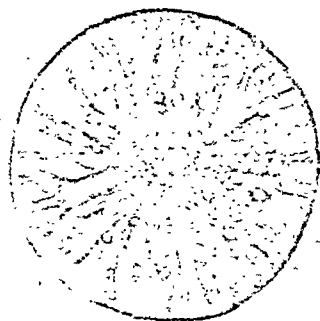
October, 1935. The narrow beam appearance of the same case as Fig. 3.

The posterior cortex gives a whitish reflex similar to, but less marked than, that found in the later stage. The difference is that no opacity can be detected by ophthalmoscopic examination.

The illustrations, Figs. 2, 3 and 4, will amplify this brief description.

Analysis of the pedigree

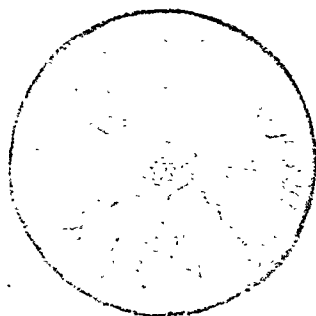
Fig. 1 has been divided into two parts, A and B, partly because the connection between the two families has not been established, and partly for convenience. Part A consists of 73 persons, 36 male and 37 female. Of the 73 there are 23 affected, 13 male and 10 female. Part B includes 58 persons, 28 male and 28 female, while the sex of two has not been ascertained. Of the 58, the number known to be affected is 17 (6 male, 11 female).



Hemlock

FIG. 2 (A.III. 14).

October 1935. The cataract is well advanced. The small picture shows the appearance by distant direct ophthalmoscopy. The larger one shows a composite slit-lamp appearance. The denser large opacities are posterior and curved like a saucer. The dots are scattered throughout the lens cortex.



Hemlock

FIG. 3 (A.IV. 17).

October, 1935. This cataract is much less advanced than that in Fig. 2. The central opacity is in the deepest layers of lens cortex, in the position shown by the interrupted white line in Fig. 4. The radiating spokes are at the same depth.



FIG. 5. (A.III. 16).

October, 1935. A peculiar pitted appearance of the central area of the retina. The round marks appear like depressions and have no resemblance to colloid or other forms of degeneration.

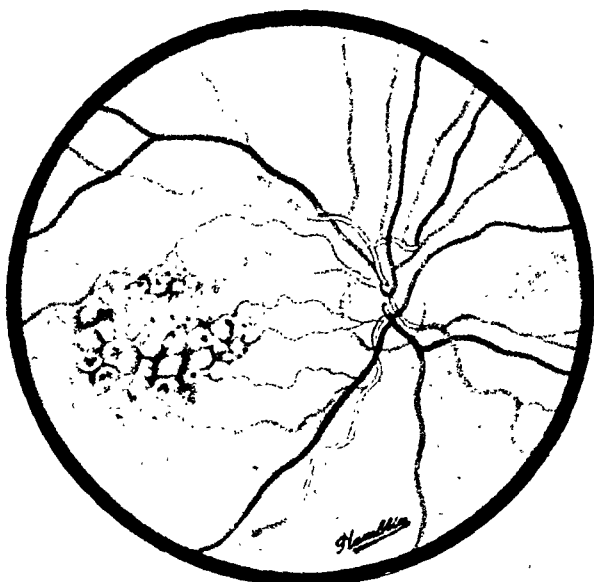


FIG. 6.

The same eye as Fig. 5, but the drawing was made in June, 1945, when the appearance was that of "Doyne's honey-comb choroiditis."

The affected persons in Part A are 31.5 per cent. of the total, in Part B 29.3 per cent. In Part A there is a slight preponderance of males among the affected members; in Part B the affected females are nearly twice as numerous as affected males. There is no particular significance in these relative figures. What is significant is that inheritance is through either sex and through affected members only. It is therefore a Mendelian dominant characteristic. Forty-one members of the family have been examined by the writer, the majority in the years 1935 and 1936. Several have been seen periodically during the last ten years and a number of these have had one or both cataracts removed during that time.

The members of the family may now be dealt with seriatim, beginning with Part A.

A.I. 1-3. Michael and Joseph Elwell were known to have had cataracts removed. Sarah (Elwell) Hedges knew that it was hereditary. She also had her cataracts removed. She was born in 1815 and died in 1892 and is well remembered by her living grandchildren.

II. 1 and III. 1, 2. Charles. Had operation. Migrated to America. Whether his two children were affected and whether they have children is not known at present.

II. 2 and III. 3, 4. Martha—unaffected and her children unaffected so far as symptoms go (not examined).

II. 3. Mary. Unmarried. Cataracts removed.

II. 4 and III. 5, 6. Unaffected and living in America.

II. 5. Phoebe Attwood (née Hedges). Cataracts removed. Was perturbed because her children were affected. Some remained single for that reason. Of her ten children eight are affected.

II. 6. David. Died nearly 70 years of age. His doctor said he had cataract but he did not have an operation. Possibly his was senile cataract. None of his children or grand-children is known to be affected. Five have been examined by the writer.

II. 7. Harry. Not affected, nor are his son or two grandsons (not examined).

II. 8. and III. 24. Unaffected and living in America (Rhode Island).

II. 9. Sarah. Unaffected. Did not marry.

III. 7. Harry Attwood. Operated. All his five children affected. The two youngest have the earliest signs yet observed, visible only with slit-lamp.

III. 8. Jessie. Unaffected and her children unaffected.

III. 9. Clara. Unmarried. Giddiness on wearing cataract glass after operation. Difficulty disappeared after gall bladder operation.

III. 14. Sidney. Operation by G. F. Haycraft 1936. Was able to work till the cataracts were far advanced (Fig. 2).

III. 16. Ada. Unmarried. Unaffected. Had a very unusual fundus abnormality. In 1935 the central area of the retina had the appearance of numerous small round depressions as if it had been struck by pellets from a shot gun, or they may have been elevations like bubbles. The colour was that of normal retina. (Fig. 5). In April, 1945, the condition had quite altered. It is not unlike Doyne's familial honeycomb choroiditis (Fig. 6).

IV. 3. Frank, aged 32 years. Served in recent war but was discharged on account of failing vision. Left cataract removed in 1943. Right developed rapidly and was removed in 1945.

IV. 4, 5. Florence and Miriam. No ophthalmoscopic sign of cataract but the posterior cortex reflects the slit-lamp beam as effectively as a developed opacity. The glow is white.

IV. 7. Not affected. Has blue sclerotics and hare lip.

IV. 10. Right cataract removed in 1944, left in 1946.

IV. 12. One cataract (? left), removed at Northampton 1942.

IV. 13-15. V. 4, 5. All living at Newport, Mon. and not examined.

IV. 17. George Attwood, aged 27 years. Affected. There has been but slight advance in his cataracts in the last ten years (Figs. 3 and 4).

V. 2, 3. These two children developed maculo-cerebral degeneration and are the subjects of a previous report (Johnstone, 1938). The elder died in February, 1943, aged 18 years. The younger died in December, 1945, aged 19 years.

PART B

B.I. 1. Benjamin Elwell, born 1807. Had one cataract removed and lost the eye. Refused operation on the other eye. His grandson (III. 10), used to lead him about Sedgley streets. His relationship to his contemporary Elwells (A.I. 1-3) has not been established, possibly cousins.

II. 5. Elizabeth Williams (née Elwell). Herself and five of her eleven children affected.

III. 10. Benjamin Russell. Belongs to an unaffected branch. His knowledge of the family has been invaluable in the investigation. He was interviewed in 1938. No attempt was made to chart the unaffected branch of the family any further than his generation as the members were somewhat scattered, many of them dead, and he knew of no cataract among them.

III. 14, 15. Both affected and both died without issue.

III. 16. IV. 1. Cataracts removed at Manchester.

III. 18. Louisa Lowndes (née Williams). Herself and four of her five children affected.

IV. 4. Joseph Lowndes, aged 45 years. Left cataract extracted by Bernard Cridland in 1931, the right by the writer 1942. Passed A.1 for Army in 1916, obtained marksman's proficiency at musketry and went overseas in October, 1916. Became a mustard gas casualty on November 9, 1918, but no after-effects. His interest and co-operation have made the investigation of Part B of the family possible. He is still following every available opportunity to extend his knowledge of the family. He is a skilled workman and foreman in the engineering trade and apparently was not

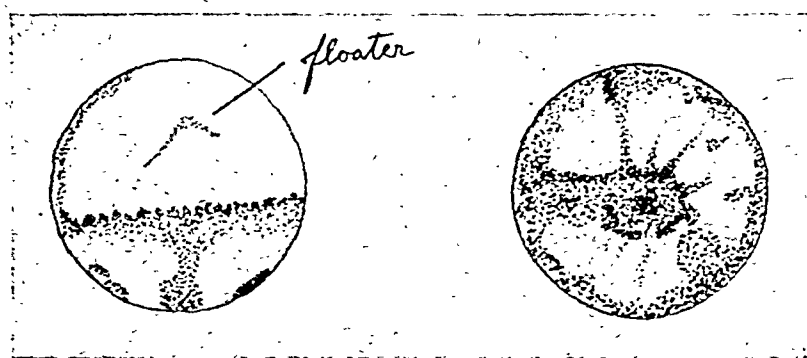


FIG. 7.

Subjective appearance in the eyepiece of a microscope drawn by Joseph Lowndes (B.IV. 4). R. cataract. L. aphakia with some capsule remnants. A vitreous opacity is shown in the upper part of field. Drawn June, 1941, a year before right cataract extension.

handicapped while using one aphakic eye during at least seven years when the right cataract was developing. In 1941 he prepared a drawing showing the shadow of his right cataract seen subjectively when looking into a microscope (Fig. 7). With it is shown the shadow from capsule in his left eye and also a vitreous opacity.

IV. 5. Beatrice. Both eyes operated on in 1918 by Bernard Cridland.

IV. 6. May. Awaiting operation now.

IV. 7. Harry, aged 39 years. Left extraction by Bernard Cridland about 1914, right by the writer in 1945.

V. 2. Dennis, aged 19 years. Both cataracts extracted by the writer.

V. 5. Gordon Stones. 1937 (aged 9 years) no sign of cataract. 1945 early signs present with posterior reflex and a few flakes.

V. 6. Graham, aged 6 years. Examined January, 1945. Left cataract with feathery posterior pattern and many scattered dots. Right less advanced.

V. 7. Vivienne (born March 15, 1943). No obvious opacity. Examined without mydriatic and without slit-lamp.

TABLE I.

	Date of Test	Serum Calcium	Serum Cholesterol	Serum Phosphorus	Serum Protein		
					Total	Albumin	Globulin
Normal	—	9-11	100-200	2-4	5-8	3-5	1-2.5
A III 9	Sept. 1946	8.1	227	—	—	—	—
A IV 3	Sept. 1945	10.0	not done	not done	7.96	5.67	2.29
A IV 10	Aug. 1946	7.14	262	3.9	—	—	—
A IV 17	April 1946	12.0	80	3.6	8.46	5.86	2.60
B IV 7	June 1945	12.0	65	3.2	7.30	3.41	3.79
B V 2	April 1946	12.0	65	3.6	7.10	5.67	1.43
A. L.	Sept. 1946	10.2	174	2.7	—	—	—
E. G.	July 1946	11.4	174	—	—	—	—

All figures are given in mg. per cent.

In Table I the summary is given of all blood estimations so far carried out. They have all been done in the last two years and the number is still small. The last two cases, A. L. and E. G., have familial cataract but do not live in the Dudley area and their connection with the main pedigree has not been traced. Serum cholesterol varies considerably in the series—two are above, three below and two are within normal limits. In the case of calcium, four are above, two slightly below and two within normal. The serum phosphorus and protein are normal in all the cases in which they were estimated.

Discussion of some published papers

In the first decade of this century, particularly from 1906 to 1910, much interest was shown in the genetics of transmissible eye defects. Nettleship and Ogilvie (1906) published the pedigree

of the Coppock family of Headington Quarry, Oxford. This family and cataract are widely known to ophthalmologists, having been discovered by Doyne in 1888 at the Oxford Eye Hospital. Adams continued Doyne's interest in the family so that few who have taken the course for the Oxford Diploma in Ophthalmology or attended the Oxford Congress, can fail to have some acquaintance with the "Coppock cataract," or "Doyne's discoid cataract." Adams (1942) has brought the story up to date with a description of the slit-lamp appearance. A point of minor interest in Nettleship and Ogilvie's paper (1906) is that the obtaining of the full genealogy of the Coppock family was entrusted to the Vicar of Headington Quarry, Johnstone by name. Doyne's discoid cataract is quite definitely congenital, the opacity involving some part of the lens nucleus and being present at birth. It is transmitted as a Mendelian dominant.

Nettleship (1909) published seven more genealogies of cataract, one of which was congenital (Case 1, Everett), one was senile (Case 4), three were possibly familial (Case 2, Perrin), (Case 3, Tomes), (Case 6, Deasley), and two were very likely familial (Case 5, Hiblen), (Case 7, Oldfield). The same author (1912) published a further case of presenile cataract.

Coming to more recent literature on the subject—Veil and Favoury (1930) present a case of probable Mendelian dominant inheritance. The pedigree is far from complete and the cataract is described as "*cataracte nucléo-corticale postérieure nummulaire avec disques stratifiés, entourés d'un halo poussiéreux.*" Like the subject of this paper, there is accentuation of the anterior capsule shagreen, some dots in the anterior cortex and the main opacity is posterior, but there the similarity ends. The chief opacity is from behind the foetal nucleus into the posterior cortex. It presents a stratified structure in concentric lamellae, indented and fitting into one another, the whole resembling a piece of money centred on the axis of the lens. It is surrounded by a ring of dust-like opacity affecting the posterior capsule, the periphery of which is transparent.

Weill and Nordmann (1930) have collected a series of six cases of endocrine disturbance with lens opacities. They divide them into two groups, (a) posterior saucer-shaped and (b) punctate and flocculent. In the first group there are three cases. Two are due to parathyroid deficiency and agree with this condition. One is a severe case of diabetes, aged 38 years. There are fine dots and some red and green crystals at the anterior surface of disjunction in addition to the posterior "*cataracte en soucoupe.*" The description suggests the picture of the cataract of this paper but the drawings are not like it at all. In group (b) two cases of myotonic

dystrophy present features resembling the present series more nearly. The third case has all the appearance of a lamellar cataract with riders; serum calcium below normal, Chvostek's sign positive and history of convulsions at age two. There is nothing in the genealogy of this present report to compare with the endocrine disorders of the six cases above.

Two papers by Caughey (1933) and Souter (1933) deal with cataract in dystrophia myotonica. The cataract in these cases has much in common with the cataract in this paper but my cases have no suggestion of muscular dystrophy or endocrine disorder. For a fuller appreciation of cataract in myotonic dystrophy the reader is referred to the above papers and to articles by Adie and Greenfield (1923) and Goulden (1928), quoted by these authors.

The purpose in referring to these cases from the literature is to draw attention to the points of similarity between cataracts associated with endocrine and metabolic disorders and my own cases without such disorders. When more is known about genetics and about endocrinology it may be this similarity will be explicable. For the present there seems to me no alternative to the view that the cataract of this paper is an hereditary feature transmitted by an autosome. As such it is not a defect or degenerative tendency on the part of the lens or the endocrine glands, but a true inherited feature. It is not apparent at birth and so cannot be called congenital. It will resemble the affected parent later on if the infant has the autosome bearing the characteristic. It may be compared with a distinctive type of nose, for example. At birth and in infancy the nose is like any other baby's nose, but as the child grows up and approaches maturity the nose develops the parental type and the similarity is easily recognised. We may say it is a familial, an inherited characteristic, but not a congenital one.

There is one more paper to which reference must be made. Hornback and De Garis (1933) give a pedigree made up of 59 persons in four generations of whom 30 had cataract, 14 male and 16 female. The unusual feature is that two males in generation II appear to have transmitted the cataract without themselves being affected. The authors rightly argue that the inheritance is an autosomal one, and dominant. To explain how an unaffected parent could have affected offspring they postulate a dominant autosomal gene which is atypical in that it produces its effect (cataract) only under certain conditions. There seems one fatal weakness in their line of argument. They produce no evidence that the two males in question didn't have cataract, beyond the fact that they were not operated on or left no history of faulty vision. Evidence of absence of cataract in the light of present day knowledge could be accepted only after examination with the slit-lamp.

and corneal microscope. From my experience familial cataract may be present for many years without the affected person having any suspicion of it. The age at which it begins to interfere with normal work and activity depends upon whether a clear space remains in or very near the visual axis or not. Some cases have been able to go on working and reading till a marked degree of opacity is visible with the ophthalmoscope; a few are in difficulty when the opacity is comparatively small but more obstructive to direct vision. The cataract is never so visible to the casual observer as a mature senile cataract. A medical practitioner may well be excused for not being able to tell if the cataract is present in the early stages. Ophthalmic surgeons may miss the cause of failing vision in young people, as actually happened with two of the members of the family herein reported. In my opinion the view that some other factors, possibly nutritional or endocrine, predispose to the realisation of the hereditary characteristic (cataract) is not proven. The "carriers" may have had the defect.

Further study of blood chemistry and endocrine activity may throw important light on all forms of cataract including the senile variety. The case for straightforward inheritance may be strengthened, or it may diminish and even disappear in the light of new knowledge in endocrinology and biochemistry.

Summary

An extensive new pedigree of familial cataract with Mendelian dominant method of inheritance is presented and the biomicroscopic appearance of the cataract is described. The possibility of other causal factors is discussed with the help of a review of the relevant literature, and a small series of blood analyses is presented.

Acknowledgments. I am indebted to the Wolverhampton Eye Infirmary for facilities to carry out the first year of the investigation and to Mr. H. B. Salt, M.Sc., of the Pathological Service, Worcester Royal Infirmary, for the blood analyses, by kind permission of Dr. McMenemey.

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A CASE OF RECURRENT APHTHOUS UVEITIS
WITH ASSOCIATED ULCUS VULVAE
ACUTUM (LIPSCHÜTZ)*

BY

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ISOLATED cases of this rare and interesting disease have been described under a variety of names from time to time. Franceschetti has traced one description as far back as 1772; occasional cases were reported towards the end of the nineteenth century; but it is only within the past fifteen years or so that it has been definitely recognised as a distinct clinical entity. Typically, the symptoms fall into three main groups: (1) periodically recurring uveitis; (2) skin eruptions of various types, e.g., papular or papulopustular, i.e., a papule resembling that of secondary syphilis with a pustular centre; recurring erythema nodosum—tender, rose-pink nodules in the subcutaneous tissue, some of which may be haemorrhagic; and transient subcutaneous nodules not involving the skin and associated with fever; (3) aphthous ulcers in the mouth and on the genitals, genital herpes, and, more rarely, the *ulcus vulvae acutum* of Lipschütz. In the male these aphthae develop on the glans, prepuce and scrotum, as well as in the mouth.

The disease affects young persons of both sexes, usually in the decade 20 to 30, and evolves slowly, by means of relapses, over a number of years. The three groups of symptoms constituting the syndrome are not necessarily present at the same time, and, as a rule, the eye symptoms appear to develop rather late. Once attacked, however, the eyes steadily deteriorate and progress, with remissions and relapses, towards blindness. One peculiarity of the disease as it affects the eyes, which is illustrated in the following description, is an alternating incidence of relapse from one eye to the other, suggesting an allergic mechanism.

Report of a case

G. M., female, aged 22 years, married, attended my clinic at the Central Middlesex County Hospital on December 5, 1944, complaining of pain and blurred vision in the left eye of three days' duration. Three teeth, which were said to have been septic, had been extracted five weeks previously. The patient neither smokes nor drinks, has always had good health, but said that she had lost weight recently. Her weight was then 7 st., 6 lbs. During

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her husband's absence in the Army she had been employed as a clerk in a Government department. She is an only child. Her parents are alive and healthy, except that her mother is said to have a weak heart. There is no personal or family history of aphthae. R.V.=6/9; L.V.=6/36 partly. The condition of the left eye was a mild acute iritis; no posterior synechiae, but the pupil dilated unevenly under atropine; there was colloid in the A.C., but no K.P. The blood W.R. was negative. A few days later the left macula was oedematous and there appeared a small patch of cloudy exudate at the periphery below. There were now large particles and much colloid in the A.C. and two or three spots of fine K.P.

By February 6, 1945, the media were much clearer. On February 20, however, the left vitreous was more hazy. There was no fresh K.P. but a circum-corneal flush was present for the first time and a new area of oedema just above the macula, with one small round haemorrhage. Mantoux reaction one inch diameter. The vitreous gradually cleared, but was again turbid on March 27. A report from the Ear, Nose and Throat Department said that there was no nasal obstruction or discharge; some chronic inflammation of both tonsils; X-rays of sinuses clear.

During the next month, apart from the appearance of a pigmented spot in the left macula, the eye continued to improve until April 17, when Cloquet's canal became hazy. The fundal periphery, however, remained clear. The blood sedimentation rate was: 1st hour /14 mm. (normal 5-8); 2nd hour /31 mm. (normal 8-14). She was examined by Dr. Joules and the chest X-rayed. A few adhesions were reported at both right and left bases and a calcified gland at the left hilum, but there was no evidence of pulmonary tuberculosis clinically or by X-ray. All the other systems were normal.

Early in July the right eye became inflamed for the first time. R.V. had fallen to 6/24. The vitreous was dusty, with cells in the A.C., and a small area of diffuse choroiditis near the macula, which was not visible with red-free (u.v.) light. The vitreous haze already noted in the left eye was still there, also the spot of pigment at the macula. A fortnight later the right macular lesion was smaller, but there was an increased shimmering reflex in this area. Somewhat later, on July 31, the haze in the left vitreous greatly increased. The right vitreous began to improve, but the enhanced macular reflex remained. The left vitreous became gradually more turbid. Several lesions could be seen in both fundi, but there was no more K.P.

At this stage the patient was very ill and felt giddy. She complained of night sweats, and the chest and abdomen were X-rayed,

with negative results. Cerebro-spinal fluid W.R. negative. Blood: Hb. 65 per cent.; W.B.C. 13,100 per c.c.; polymorphs 55 per cent., lymphocytes 36 per cent.; monocytes 5 per cent.; eosinophils 3 per cent.; basophils 1 per cent. Urine, acid; sp.gr. 1022; nothing abnormal found. Two teeth were extracted and this was followed by a rise in temperature, $101^{\circ}2\text{F}$. During the early part of September the evening T° regularly rose to 100° , and during the latter half of the same month to 103°F . She was admitted to hospital on August 23 and treated with sulphonamides. A total of 40 grms. sulphamezathine and 35 grms. of sulphathiazole was administered without any effect.

On October 16 the left eye suffered another acute relapse. Microscopic examination of the cerebro-spinal fluid revealed a few leucocytes and occasional hyaline casts. B.S.R. 12 mm./1st hour. A fortnight later the media of the right eye, which had been steadily clearing, suddenly became dull. The right eye, however, after this temporary lapse, again began to clear, but a new lesion (choroidal—not visible with red-free light) appeared in the macular region of the left fundus. During November both eyes became noticeably clearer, until on December 11 the patient reported that the right eye had been inflamed and "black" for a week. This proved the most severe of the recurrences in the right eye. There were two spots of fresh K.P. and the media were very turbid. The disc was obscured by exudate and vitreous haze. A large grey area, apparently of exudate, was present in the region of the macula. Some new, minute spots were seen in the left macula. B.S.R. 8mm./1st hour. The media of the right eye soon began to clear, but early in January, 1946, the patient again developed a temperature and complained of aching of the left upper jaw. Five teeth were extracted, happily without any local reaction or rise of temperature.

Some time towards the end of November or beginning of December, 1945, apparently about the time of the last severe relapse in the right eye, the patient had a rigor, which, she said, lasted on and off for three days, and was sufficiently severe to cause chattering of the teeth. Rigors recurred at intervals until April, 1946, altogether three or four. During these the patient remained at home and was not seen by me or by any of the physicians. Towards the end of January, 1946, she was confined to bed at home on account of pain in the left side of the chest. This was diagnosed as influenza. When she reported at my clinic on January 29 she was re-examined by a physician who found no evidence of pleurisy. Her temperature during this period was said to have risen to 102°F . This illness ended with a recurrence, accompanied by circum-corneal injection, in the left eye. B.S.R.

25 mm./1st hour. When I saw her on February 26 the media of both eyes, particularly the right, were clearing. R.V.<6/60; L.V.=6/60.

On this date (February 26, 1946) the patient reported for the first time a small vulval sore, situated on the left labium, which

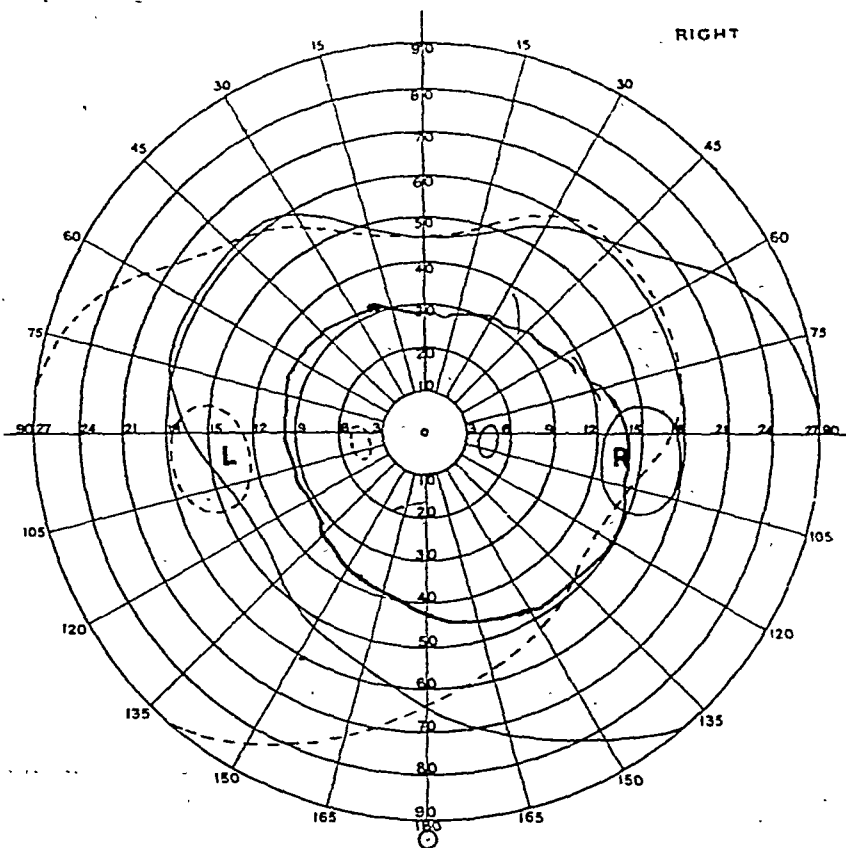


FIG. 1.

This chart, although taken in January, 1947, is representative of a series taken of the right eye during 1946. The object was a 3mm. white disk. It represents the peripheral limits of the field only, as it was found impossible to plot the irregular distribution of patches and gradients of vision and blindness which constitute the actual field.

was painful for five days but was now healed. At the same time there were small ulcers in the mouth. There were three or four of these, one on the left edge of the tongue and the others inside the cheek and inside the lower lip. The ulcers were roughly oval in shape, clean, shallow, and with sharply defined edges, approximately 2-5 mm. in length. A fortnight later the patient had a

severe uterine haemorrhage with clots and was pale but not collapsed. She was readmitted to hospital under a gynaecologist.

On examination no pelvic inflammation was found. By this time the patient's husband had been demobilised, but the patient denied the possibility of pregnancy and miscarriage. Subsequent events, however, suggest that this was the explanation. There was no vulval ulceration at this time, but the gynaecologist reported a "peculiar condition of the vaginal mucosa." The vagina was granular and tender, and the mucosa of the vault hard, rough and irregular. B.S.R. 15 mm./1st hour. After a few days in hospital she was discharged.

She was readmitted on April 3 for further investigation. She now complained of tiredness, night sweats, loss of weight, and a recurrence in the right eye. There had been a recent rigor. The right eye had one posterior synechia and the vitreous was very turbid. Throughout her stay the evening temperature was $99^{\circ}2$. The right tonsillar gland was slightly enlarged, but the tonsils were small and were reported normal. Clinical examination of the chest and X-ray examination negative. Hb. 100 per cent.; W.B.C. 9,800; reds 4,250,000; platelets 425,000 approx; bleeding-time two min.; clotting-time three min., 20 sec. A catheter specimen of urine showed occasional leucocytes and reds; no organisms in a Gram film, and culture sterile. Throat swab—no haemolytic streptococci. Serum failed to agglutinate *Brucella abortus*. There were 31 units antistreptolysin "O" present per c.c., which is within normal limits. Mantoux 1:10,000++. Capillary resistance test negative. A complete clinical examination was made with negative results, but five teeth were regarded as suspicious and were extracted. The socket of one of these was septic for some time after extraction. There was one small ulcer present on the tip of the tongue.

On May 6, 1946, the left eye flared up again. The pupil did not dilate and there was yellowish discolouration of the iris, with much colloid in the A.C. and corneal epithelial oedema. The tension of the left eye was soft. Owing to the corneal oedema and turbidity of the vitreous, the pupillary red reflex was poor, but a yellowish grey mass could be detected in the upper nasal region of the posterior vitreous. The right eye had not changed; there were still massive vitreous opacities and also the lesion at the macula already noted. For some weeks both eyes remained more or less in this condition, except that the red reflex improved somewhat in the left eye, although the mass in the vitreous was still present.

The patient was admitted to the Western Ophthalmic Hospital on July 12. The Mantoux reaction was over three inches in dia-

meter on the third day, and it was decided to give non-specific protein therapy. Two intra-muscular injections of milk (10 c.c. each) were followed by four intravenous TAB injections, the initial dose being 20 million B. Typhosus, 10 million B. Paratyph. A, and 10 million B. Para B. The right eye cleared very considerably and even the left eye began to show a brighter red reflex surrounding the exudation in the vitreous. This seemed to me the first response to treatment so far attained. Further, the fairly regular alternation of individual relapses, first in one eye, followed by an improvement, next in the other, had suggested to me for some time that there must be an allergic mechanism present, *i.e.*, an attack in one eye sensitising the uvea of the other and inducing a recurrence, which in turn became the excitator, and so on, in cyclic fashion. As I still suspected an underlying tuberculous infection I decided to attempt desensitisation by means of tuberculin injections. Accordingly, the patient was given weekly subcutaneous injections of old tuberculin, beginning with 1/1,000,000 mgrms., and very slightly and gradually increasing the dose. The improvement continued in both eyes. The right vitreous became very clear, apart from one or two moderately large opacities, and even the left seemed to be clearing satisfactorily, although the pupil remained irregular, the tension somewhat soft, and the A.C. shallow. The patient's general health had improved. The B.P. registered 120/60. About this time the patient announced that she was pregnant and this was confirmed by Mr. MacVine.

On October 16 the patient reported that she had noticed vulval ulceration for the past two months, and Mr. MacVine reported a small, rather painful ulcer situated on the left labium, for which he prescribed a 2 per cent. gentian violet paint. Within a month two ulcers developed at the same site and a contact ulcer, opposite the first, on the right side. -The contact ulcer, however, rapidly healed and has not recurred. The original ulcer of the left pair grew rapidly, and was indurated, with raised edges, and a base composed of a thin layer of firm, slightly nodular, pale granulations, covered by a thin, light-coloured film. Mr. MacVine performed a biopsy, removing a wedge through the base of the ulcer, and inserting four catgut sutures. Dr. W. Pagel reported on the section as follows: "the ulcer goes fairly deep into the submucosa, its floor being formed almost completely by polymorphs. There is no clue as to the aetiology in the section examined. In the periphery of the ulcer the cells are chiefly lymphocytic: there are also some eosinophils and plasma cells. We are preparing a Ziehl-Nielsen section but will not report unless acid-fast elements are found." B.P. 136/70; B.S.R. 16 mm./1st

hour. Penicillin was administered locally and systemically, a total of 735,000 units, with no response. Later, the vulval ulcer was treated with a concentrated solution of urea. At the same time small ulcers, some with a sloughy base, appeared on the tongue and lips, especially on the right edge of the tongue. Culture from these yielded a few colonies of streptococcus viridans.

Just before treatment with penicillin, a rhinological report described some thickening of the lining membrane of both antra, particularly the left, and pronounced the frontal and posterior sinuses, and the posterior ethmoidal cells as normal. The hands were again examined by X-rays for cystic changes and were found normal. Analysis of the cerebro-spinal fluid gave protein 20 mgrms. per cent.; no excess of globulin; chlorides 720 mgrms. per cent.; W.B.C. 1 per cent. c.mm.; R.B.C. 250 per c.mm. The pressure was normal, and the Lange curve 0012110000. The W.R. was negative, and the complement fixation test (blood-serum) for gonorrhoea also negative.

It had now become clear that the diagnosis of this condition was the syndrome which is, perhaps, best described by the term recurrent aphthous uveitis, and that the vulval ulcer was the *ulcus vulvae acutum* of Lipschütz. Both eyes were still fairly quiet, the vulval ulcer very painful, and the aphthae in the mouth troublesome. On December 7, however, the left eye suddenly flared up and became quite blind. A necrotic, apparently encapsuled exudation occupied the whole of the posterior vitreous, giving rise to the amaurotic cat's eye reflex of Beer, and pressed forwards the lens, which remained clear, so that the A.C. was nearly obliterated and the pupil semi-dilated and fixed. A large emergent conjunctival vessel developed on the temporal side in the position of the ciliary body. The right fundus, on examination with red-free (u.v.) light, showed pale spots and streaks in the macular area, evidence of early micro-cystic degeneration of the retina. The optic disc looked very yellow—the patient is a blonde. There was a moderate amount of aqueous flare in the right A.C. and a large amount in the left. The larger of the vulval ulcers showed signs of healing, forming a waist across its centre. A further recurrence in the right eye was noted on February 24 and the media again became very turbid for some days. There were no ulcers in the mouth—the last was on February 18. Just before this last attack in the right eye the vision with correction (0.5 cyl. axis 150°) = 6/36.

At first the foetal presentation was a breech, but towards the end of pregnancy it corrected itself. Mr. MacVine performed a Caesarian operation on March 6 and extracted a female child, weighing just over 5 lbs. but otherwise normal. During the night



FIG. 2.

Ulcus vulvae acutum of Lipschütz. When the drawing was made the ulcer had begun to heal. Its maximum dimensions were 23mm. by 20mm. In the figure the left thigh is flexed and abducted in order to expose the ulcer fully.

of March 9 the patient had a severe rigor, accompanied by a temperature of $105^{\circ}2$ F., and two more next day. Afterwards convalescence proceeded normally and the patient was discharged from hospital on March 26. A very mild and transient relapse occurred in the right eye shortly after the last rigor, and on March 19 the left eye became painful, interfering with sleep for about three days. On March 18 both vulval ulcers had completely healed with a normal and perfectly healthy scar. The patient felt and appeared to be remarkably well. A curious and perhaps significant point in connection with the vulval ulcers is that from first to



FIG. 3.

Microscopic section of *ulcus vulvae acutum*. Low power. Material from biopsy. Cell infiltration mainly lymphocytic but also with a good many plasma cells. Vessels typical of granulation tissue.

last, covering eight or nine months, there was no enlargement of the inguinal glands. Moreover, the second group of symptoms characteristic of this syndrome, *viz.*, skin lesions and eruptions, has been entirely absent. The patient has a fine, healthy skin, above the average, and at no time has there been a trace of papules, spots or other lesion.

Comments

I have given the history of this case in detail and at length, perhaps in too much detail and at too great length, but in describing an obscure and apparently rare disease—one which, so far as I know, has not been described before in British ophthalmological literature—with an unknown aetiology, it is impossible to foresee which of its manifestations will prove to be relevant in the light



FIG. 4.

The same as Fig. 3. Margin of the ulcer showing the epithelialized edge.

of fuller knowledge. During the long course of the disease my own attitude to its probable aetiology passed through three stages: (1) the focal sepsis phase; (2) the tuberculous phase; (3) the viral phase. The initial attack of iritis, which ushered in the disease, appeared then to be the ordinary iritis or iridocyclitis that is not uncommon in young adults. A history of tooth extraction a short time previously seemed to confirm this assumption, and further investigations were planned on this hypothesis, which was not shaken by finding a rather high blood sedimentation rate. Before



FIG. 5.

The same as Fig. 3. High power.

long, however, the loss of weight, night sweats and pyrexia, associated with a pronounced Mantoux reaction and high B.S.R., especially after the second eye had become involved, led me to suspect, even after clinical examination and X-rays of the chest had proved negative, an occult tuberculous infection. As the disease progressed, the apparent alternation of attacks from one eye to the other, lent colour to this suspicion, as it is generally agreed that sensitisation of tissues by tuberculous toxins explains some of its phenomena. The following table shows the dates of incidence from the beginning up to the time of writing :

TABLE I

1944	1945						1946				1947		
L. 2/12	20/2	—	31/7	16/10	—	—	29/1	—	7/5	7/12	—	—	19/3
R.	—	5/7	—	—	30/10	11/12	—	3/4	—	—	24/2	10/3	—

Altogether there have been fourteen attacks, eight in the left eye and six in the right. The series does not show exact alternation, but it is probably sufficiently close to conform with the theory of sensitisation. It may be remarked too that certain observers, *e.g.*, Stahli (1922), Urbanek (1934), and Meller (1934), who have reported cases of this disease, have advocated a tuberculous aetiology.

For over a year there were no aphthae in the mouth, nor as far as I knew, on the genitals. When buccal aphthae did appear, not sufficient attention was paid to them as the physician who was then in charge regarded them as dyspeptic ulcers. It must be remembered, also, that the teeth gave trouble on several occasions and some were extracted. This was a mistake of which I was not cognisant at the time, but, fortunately, there is no evidence that it provoked a focal reaction. The buccal ulcers, however, were at first more or less unconsciously associated with the condition of the teeth.

A remarkable feature of the relapses was their sudden onset and relatively short duration. Without any warning, as it were, an eye became affected acutely overnight, reaching its maximum intensity all at once, and then declining in a day or two. This also must be regarded as compatible with an allergic reaction, and I think it must be accepted that allergy does play a part in the symptomatology of this disease, although, as Franceschetti remarks, of a secondary character. Hypopyon, a characteristic feature of early descriptions, apparently of a transient character,

appearing and reappearing many times in the course of a single day, was never present in this case.

It was not until vulval ulcers appeared, some eighteen months after the initial attack in the left eye—apparently one or perhaps two vulval sores developed six months earlier, but were not reported by the patient until later—that the true nature of the condition became evident. The striking and very intractable ulcer, which developed on the labium majus, and proved absolutely resistant to every form of local treatment applied, was then recognised as identical with that described by Lipschütz in 1913 under the name *ulcus vulvae acutum*. Complete indifference to treatment, indeed, is the predominant feature of this disease. At one time non-specific protein therapy seemed to produce a favourable result. The patient herself was convinced of this, but the explanation of the apparent improvement then observed may quite well be, and probably is, that on this occasion the treatment coincided by chance with the beginning of a period of remission.

While Weekers and Reginster, in their second paper (1938), were the first to stress the independent nature of this disease, the credit for its complete elucidation belongs, in the first place, to Touraine (1941) and to some extent to Dascalopoulos (1932)—*l'uvéïte récidivante aphteuse*—and, secondly, to Hulusi Bêhçet (1937) and to Franceschetti and Valerio (1939), who first advocated the theory of a viral aetiology. In support of this theory Franceschetti, Valerio and Babel (1946) state that recurrent aphthous uveitis has some resemblance to periodic ophthalmia in horses, which, however, has not been certainly proved to be a viral infection, and also to bovine aphthous fever, which is caused by a known virus transmissible to man.

It is not improbable that the pathogenic agents known as filtrable vira play a more important part in the causation of intra-ocular infections, *e.g.*, sympathetic ophthalmitis, also a disease of the young, associated with injury and resistant to treatment, than is commonly supposed. Vira vary in size; the largest are almost as big as the smallest bacteria; the smallest approach the size of large molecules. According to one school of thought the virus is a living organism; according to another it is an enzyme. Vira, however, exhibit the phenomenon of variation, which is a characteristic of living matter but not of enzymes. Essentially they are cell rather than body infections, as they appear to lack the enzymes necessary for independent growth. This peculiarity may explain their immunity from the effect of drugs. In other words, they are intra-cellular parasites, and in many diseases "inclusion bodies" are seen in the infected cells, *e.g.*, the

Guarneri bodies of smallpox; Negri bodies of rabies; Prowazek-Halberstaedter inclusions of trachoma, etc. They attack the cells of all three germinal layers, with the exception of muscle cells, and tend to infect young, healthy cells and young, healthy individuals rather than old, unhealthy ones. Rivers has explained that the part played by injury in causing viral lesions is due to the presence of young cells participating in the process of repair. For the same reason, embryonic cells are the best nutrient medium for their cultivation. The viral diseases constitute a large group of diverse conditions, such as measles, poliomyelitis, yellow fever, coryza, Rous' fowl sarcoma, etc. As a rule, one attack confers a life-long immunity, but influenza, the common cold, and herpes simplex are exceptions. They do not respond to sulphonamides, penicillin, or to any other known agent. Professor Bêhçet and his colleague Professor Braun, of the University of Istanbul, claim to have demonstrated inclusion bodies, or rather, the "elementary bodies," of which the cell inclusions are composed, in smears from the mouth and genital lesions of two cases. The smears were stained by the Giemsa and Herzeberg methods and formations resembling the elementary bodies of viral disease were found with each recurrence. Three patients, who also had aphthous ulcers of the mouth or genitals, were used as controls, and smears examined. Bêhçet and Braun, however, failed to find these bodies in the material from the controls. When stained with Giemsa, the bodies are approximately the same size as Guarneri bodies, *i.e.*, extremely small and round, reddish purple in colour, partly intra-cellular and partly extra-cellular. The same bodies were found after staining with Victoria-blue by the Herzeberg method.

These findings were observed on three different occasions and experiments were devised to determine whether they were artefacts or not. For example, smears dried in air and treated with alcohol-ether in order to extract any lipoids present, then stained with Giemsa and Herzeberg, showed abundance of these bodies. Also when treated with 3 per cent. acetic acid solution for fifteen minutes and stained, the picture was the same. After treatment with 3 per cent. sodium hydroxide solution for fifteen minutes, however, these bodies were no longer found. Smears taken from the vulval ulcer in my case by Dr. J. D. A. Gray and examined by Dr. F. O. MacCallum of the Wellcome Bureau of Scientific Research, proved negative. It should be noted, however, that when these smears were taken the ulcer was healing rapidly and for the present the negative result must be regarded as inconclusive.

Finally, attention should be drawn to one or two unusual features in my case. First, the fact of pregnancy. It is difficult even now to judge whether pregnancy and the metabolic changes

associated with it affected the course of the disease one way or the other. It is true that about the sixth month of pregnancy the left eye suffered its most severe attack and became blind, and that for the greater part of the period the vulval ulcer, not only showed no signs of healing, but became gradually worse, yet the general impression in my mind was that on the whole the effect was favourable. The ulcer began to heal during the last month or two of gestation and healed very rapidly after delivery. It may be recalled that there was a probable earlier miscarriage. Aphthae appeared from time to time during pregnancy. It is interesting to note here an observation by a colleague, *viz.*, that his wife who is subject to buccal aphthae, without other symptoms, during her pregnancies has always been free. The pregnancy of my patient was attended throughout by no untoward symptoms or discomfort.

Secondly, at one stage, lasting about five months, there occurred three or four severe and inexplicable rigors and again three days after delivery three rigors, apparently unconnected with the Caesarian section, for there was no evidence of uterine infection, followed one another within a period of about twelve hours. Otherwise, convalescence was uneventful.

Attention has already been called to the entire absence of skin symptoms; to the absence of leucocytosis; and to the absence of response to penicillin, sulphonamides, and other forms of treatment. Taken as a whole, the evidence, some positive and some negative, seems to point to infection by a virus as the most probable explanation of the nature of this disease.

Summary

The history of a case of recurrent aphthous uveitis is described over a period of twenty-eight months.

There were no dermatological symptoms but the *ulcus vulvae acutum* of Lipschütz was present.

Pregnancy occurred and went on to term.

Rigors occurred on several occasions.

Resistance to treatment is emphasised.

The hypothesis of a viral infection is discussed.

I have pleasure in expressing my thanks to Dr. Joules, the Medical Director, and to other members of the staff of the Central Middlesex County Hospital, especially Mr. MacVine, Dr. W. Pagel and Dr. J. D. A. Gray, for their invaluable help and co-operation.

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FURTHER EXPERIENCE WITH AMNIOTIC MEMBRANE GRAFTS IN CAUSTIC BURNS OF THE EYE*

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In an earlier communication (Sorsby and Symons, 1946) attention was drawn to the value of amniotic membrane grafts in the treatment of caustic burns of the eye. In a series of 30 cases (the burn being produced by lime in 22 cases; by hydrochloric acid, sodium hydroxide, and ammonia in 2 cases each; and by liquid sulphur dioxide and lead in 1 case each) no corneal or conjunctiva sequelae were noted in 27 cases. In 28 eyes in which vision was known 26 showed vision of 6/9-6/5. A method of applying the graft was described and it was indicated that early grafting gives the best results. Generally speaking the eyes were found to be very nearly white in 3 to 5 days after grafting and they were usually normal by the end of a week.

1.—A further series of 28 cases

Table I sets out the distribution and the clinical details in a further series of 28 cases. These fall into three distinct groups:

* Received for publication, May 5, 1947.

1. LIME BURNS (21 cases).

(a) *Severity*.—The severity of the burns can be judged by the following data :

Conjunctiva :

Both upper and lower fornices burnt	3
Lower fornix only	16
Inner half of conjunctiva	1
Lower fornix and outer half of conjunctiva ...	1

Cornea :

Clear	1
Whole of cornea involved	2
Three quarters of cornea involved	3
Half of cornea involved	6
Lesser areas of cornea involved	9

(b) *Time of Grafting*.—Grafts were invariably applied on the same day as the patient was seen. In 9 cases this corresponded to the day of the burn; in 10 cases it was a day after the injury was sustained. In one case the patient was not seen until 2 days after the injury, and in one more until after 4 days.

(c) *Duration of Treatment*.—Eight of these 21 cases were treated as out-patients. The time taken for the eye to become normal is shown in the following summary table :

Eye normal in 2 to 5 days	10 cases
„ „ „ 6 to 8 „	7 „
„ „ „ 9 to 12 „	0 „
„ „ „ 3 to 18 „	3 „
Unrecorded	1 case

(d) *End result*. (1) *Cornea*.—In all but two cases the cornea was clear on discharge. In the two cases in which there was a corneal opacity a pre-existing corneal scar was present in one (No. 18), whilst delay in treatment appears to have been the responsible factor in the second one (No. 1), the one case in which the graft was carried out 4 days after the burn.

(2) *Vision*.—Apart from the one feature with a pre-existing corneal scar in whom vision was 6/24, vision was good, or could

be presumed good in the remaining cases. It was 6/6 to 6/5 in 12, 6/9 in 5 cases, and it was not recorded in 3 more, 2 of whom were illiterate children.

(c) *Complications and sequelae*.—Apart from the corneal opacity in case 1 already noted, a tendency to symblepharon was noted in 2 cases (Nos. 13 and 16). In each case this tendency was suppressed by grafting; in case No. 13 the one graft used was adequate, whilst in No. 16 a further graft had to be inserted after 48 hours. In two more cases a second graft was found necessary: in case 9, treated as an out-patient, the graft was found under the bandage when the eye was dressed after 48 hours; in case 19 there was the unique experience of more staining of the conjunctiva being present on removing the bandage after 48 hours; in both these cases there was an uneventful recovery after a second graft.

2. SODIUM HYDROXIDE (5 cases).

Three of the 5 burns produced by caustic soda can be dismissed briefly. The lower fornix was involved in 2 cases and the inner half of the conjunctiva in the third; the cornea was involved to the extent of $\frac{1}{4}$, $\frac{1}{2}$ and $\frac{3}{4}$ respectively. The grafts were all applied on the day of the burn. Two cases became normal in three days and one required 9 days. The cornea was clear in all cases and vision was 6/5 in one case and 6/9 with correction in the two others.

Considerably more severe injuries were noted in the two eyes of a man, aged 64 years. (Nos. 25 and 26.) When seen three hours after an explosion, in which his eyes were burned, both cornea were pearly opaque. Amniotic grafting was carried out on the same day, and subsequently twice more at intervals of 4 days, and finally once more 6 days later. A hypopyon developed in the right eye 20 days after the burn, when paracentesis and anterior chamber lavage were carried out. After prolonged treatment vision of the right eye was reduced to perception of light; a dead-white opacity of the cornea is present. In the left eye the lower two-thirds of the cornea is intensely opaque and vision is counting fingers at one metre.

3. FIREWORKS (2 cases).

Of the two injuries produced by fireworks one was a superficial conjunctival burn on the left side with three-quarters of the cornea staining. There was no conjunctival burn on the right side, but one-eighth of the cornea showed staining. Amniotic membrane grafting was carried out on the left eye on the day of injury with an excellent result.

TABLE I
Clinical details on caustic burns treated by amniotic graft.

No.	Age	Extent of cornea involved	Conjunctival forncies involved	No. of days grafted after burn	No. of days to become normal after grafting	END RESULT		Remarks
						State of cornea	Vision (with correction where applicable)	
						1. LIME BURNS		
1	28	1/4	Lower	4	17	Scar	6/9	First seen 4 days after injury.
2	30	1/4	Lower	0	5	Clear	6/6	—
3	19	1/4	Lower	1	2	Clear	6/5	Out-patient.
4	30	3/4	Upper and lower	1	4	Clear	6/6	—
5	16	1/4	Lower	0	6	Clear	6/6	—
6	42	3/4	Lower	1	8	Clear	6/6	—
7	23	1/4	Lower	0	3	Clear	6/5	Out-patient.
8	51	1/4	Lower	0	5	Clear	6/6	—
9	32	1/4	Lower	1	5	Clear	Not recorded 6/9	Two grafts. Out-patient.
10	17	1/4	Lower	1	5	Clear		

TABLE 1—continued.
Clinical details on caustic burns treated by amniotic graft.

No.	Age	Extent of cornea involved	Conjunctival forfices involved	No. of day grafted after burn	No. of days to become normal after grafting	END RESULT		Remarks
						State of cornea	Vision (with correction where applicable)	
11	33	1/2	Lower	0	6	Clear	6/9	Out-patient.
12	52	Complete	Upper and lower	0	Not recorded	Clear	6/9	Out-patient.
13	13	None	Lower	1	5	Clear	6/6	Tendency to symblepharon suppressed by grafting.
14	33	1/2	None	1	3	Clear	6/5	Inner half of bulbar conjunctiva involved.
15	6	1/2	Lower	1	3	Clear	Not recorded	Outer half of bulbar conjunctiva also involved.
16	6	1/2	Lower	0	7	Clear	Not recorded	Tendency to symblepharon suppressed by a second graft.
17	30	1/2	Lower	1	6	Clear	6/6	Out-patient.
18	6+	1/6	Lower	2	13	Old scar	6/2+	Out-patient. Old corneal nebula.
19	31	1/2	Lower	0	8	Clear	6/6	Out-patient. Two grafts. More staining after first graft.
20	53	1/2	Lower	1	7	Clear	6/6	—

TABLE I—continued.
Clinical details on caustic burns treated by amniotic graft.

No.	Age	Extent of cornea involved	Conjunctival fornices involved	No. of day grafted after burn	No. of days to become normal after grafting	END RESULT		Remarks
						State of cornea	Vision (with correction where applicable)	
21	31	Complete	Upper and lower	0	18	Clear	6/9	Central corneal opacity. Intense chemosis at first grafting. Lime found present on first dressing 2 days later; grafted again.
22	47	1/2	Lower	0	2. SODIUM HYDROXIDE		6/9	BURNS
23	33	1/2	Lower	0	3	Clear	6/9	Out-patient.
24	41	1/2	None	0	3	Clear	6/5	Inner half of bulbar conjunctiva involved.
25	64	Complete	Upper and lower	0	—	Opaque	P.L.	Extensive opacification of cornea at injury.
26						Opaque	C.F.	
27	11	Complete	Upper and lower	1	49	Opaque	C.F.	Extensive burn of cornea and sclera. Im- mediate opacification of cornea.
28	17	1/2	Lower	0	7	Clear	6/9	—

The second case was a severe burn involving the conjunctiva, episclera, sclera, and cornea of the right eye giving staining of the whole of the cornea with a total opacity. Though the final result was counting fingers in that eye, the clinical course during treatment and convalescence was not unsatisfactory. There was no marked tendency towards symblepharon, nor was there any worsening of the initial condition of the eye.

2.—Discussion

1. *Significance of early grafting.*—In the previous communication the difficulty in obtaining experimental control of the findings recorded was indicated. Nor could adequate clinical control be employed. Some approach to clinical control is, however, available by comparing the results obtained in the first series with those recorded in the present series. The 22 lime burns in the first series were submitted to grafting by amniotic membrane at variable times after the injury. At the beginning early grafting was exceptional, and it was only towards the end of the investigation that grafting on the day of the injury was undertaken. Classifying the data previously recorded the following summary table emerges.

Number of day after burn when grafting carried out.	Duration in days of in-patient treatment for each case respectively
0	5, 2, 4, and no in-patient treatment in two cases
1	5
2	10, 8, 7, 12, 10, 6, 2, 5.
3	17, 15, 11, 27
4	9
5	18
6	21
10	17

These results in themselves bear out the suggestion made in the earlier communication that early treatment is advisable. This suggestion is strengthened by the data in the following

summary table on the present series extracted from Table I to show the time taken for the eye to recover its normal state in relation to the time when the graft was applied after the burn:

Number of day after burn when grafting carried out.	Number of days for eye to become normal in each case respectively
0	5, 6, 3, 5, 6, 7, 7, 8, 18*
1	2, 4, 8, 5, 5, 5, 3, 3, 6, 7
2	13
4	17

* Case No. 21. Lime found in eye at first dressing of amniotic graft.

The fact that in both series late grafting involved prolonged treatment is indicative of the value of the procedure.

2. *Mode of Action.*—The experimental results previously reported suggested that human amniotic membrane was species specific in its action, and possibly tissue specific too, in so far as it reacted well with conjunctiva but not with skin. There is no support for species specificity or tissue specificity from experiments with tissue culture (Sorsby and Ungar, 1947). It would appear that amniotic membrane does not stimulate growth, but that it acts as a sort of internal splint for the proliferating tissue.

3. *Mode of use.*—The method previously described has stood the test of further use. Occasionally the procedure of inserting amniotic membrane has been modified in particular instances to cover defects in the conjunctiva not readily served by an implant into the fornix. In such cases partial covering of the cornea has sometimes to be used. In most cases such partial covering does not appear to act deleteriously in contrast to total covering of the cornea; moreover, the membrane generally tends to retract leaving the cornea fully exposed.

4. *Limitations of amniotic membrane grafting.*—Cases Nos. 25, 26 and 27, which illustrate immediate and severe damage to the cornea, show the limitations of amniotic membrane grafting. It is necessary to distinguish clearly between immediate (and presumably irreversible) damage to the cornea and secondary involvement of the cornea from a persistent raw conjunctiva.

Amniotic membrane grafting will have no effect on the first; it is invaluable as a preventative of the second type of corneal damage. A crude parallel is the cornea in ophthalmia neonatorum. The sulphonamides and penicillin have little effect on the damaged cornea; they are invaluable in preventing corneal ulceration and infection.

5. *Method of preparing amniotic membrane grafts.*—We are indebted to Mr. H. P. Morley of the Ligature Department, The London Hospital, for the following note on the mode of preparing amniotic membrane grafts of human origin. In his view the method of preparation is sufficiently simple to be possible in any well equipped pathological laboratory or dispensary.

(1) The placenta should be placed in normal saline immediately after delivery, the following process begun within twenty-four hours.

(2) Carefully separate the amnion from the chorion, commencing from the edge of the fringe up to the cord.

(3) Wash the amnion thoroughly in running water, then place in a saturated solution of common salt for twelve hours.

(4) Remove the amnion and immerse in a fresh saturated solution of common salt for twelve hours.

(5) Extend the amnion on black-backed plate glass and remove mucus and surface fat with gauze swabs.

(6) Wash thoroughly until the membrane is free from salt and immerse in distilled water from some ten to thirty minutes, frequently agitating the solution.

(7) Immerse the amnion in a 1 in 150 solution of potassium hydroxide (caustic potash) frequently agitating for at least one hour. No membrane being constant, the time factor is governed by the appearance of the membrane and experience.

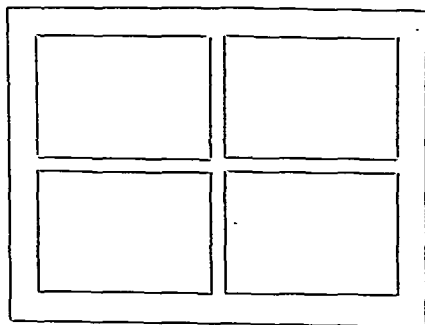
(8) Extend the membrane on the plate glass and wash with distilled water, removing soaps formed.

(9) Pour hydroxide solution on the membrane and remove all remaining fat by manipulative massage with gauze swab. If necessary, further hydroxide baths and manipulative massage should be applied.

(10) Wash the membrane free of alkali.

(11) Test for fat by re-saponification.

(12) Immerse the membrane for twelve hours in distilled water, then stretch on wood frames measuring—outside measurements— $6\frac{1}{2}$ in. \times $8\frac{1}{2}$ in., divided into four sections. The $6\frac{1}{2}$ in. \times $8\frac{1}{2}$ in. frames can be made of smooth, polished wood measuring $\frac{1}{2}$ in. \times $\frac{1}{2}$ in. and the wood dividing the frame into four, $\frac{1}{4}$ in. wide by $\frac{1}{2}$ in. deep.



Drawn to $\frac{1}{4}$ scale.

The membrane will adhere naturally to the frames.

(13) Allow to dry at ordinary room temperature, then remove from the frame and trim.

(14) Sterilisation is effected by dry heat at 150°C. for one hour in open-mouthed cellophane envelopes.

Summary

(1) A further series of 28 cases of caustic burns of the eye treated by grafting with human amniotic membrane is recorded.

(2) There were 21 cases of lime burns. In 10 of these the eye was normal within 2 to 5 days, and in 7 more within 6 to 8 days. In all cases but one the cornea was clear at the end of treatment. Vision of 6/9-6/5 was recorded in 17 cases, and could be presumed in three more. In two cases a tendency to symblepharon was controlled by grafting.

(3) There were 5 cases of burn due to sodium hydroxide, and 2 to fireworks. In 3 of these 7 eyes there was severe immediate corneal damage. This was uninfluenced by amniotic membrane grafting, though grafting affected favourably the course of these eyes.

(4) Taking the previous and the present series together it is shown that remarkably rapid response may be expected when grafting is applied on the same day, or within 24 hours after the injury; subsequently, recovery is slower.

(5) The mode of action of amniotic membrane grafting is not clear. It would appear that the amniotic membrane does not stimulate growth, but acts as a sort of internal splint for the proliferating tissue.

(6) The method of preparing amniotic membrane, as practised by Mr. H. P. Morley, at the London Hospital, is described.

We are indebted to Sister Hollands and Sister Thorogood of the Ophthalmic Unit at Lambeth L.C.C. Hospital for their help with the treatment of these cases.

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VISUAL DIAGNOSIS OF EYE DISEASES BY
MEANS OF INFRA-RED RADIATION¹

BY

A. VAŠKO and M. PELEŠKA

PRAGUE

LATELY we have been experimenting with image converters² which are sensitive to infra-red radiation in an attempt to aid the diagnosis of certain diseases of the eye. We are studying normal as well as pathological tissues of the anterior and posterior parts of the eye and also investigating the possibility of seeing through opacities of all the component parts of the eye (cornea, aqueous humour, lens and vitreous humour).

For this purpose we measured the spectral transmission characteristics of these media. Fig. 1 shows the spectral distribution of transparence of a very opaque cornea and lens, the former

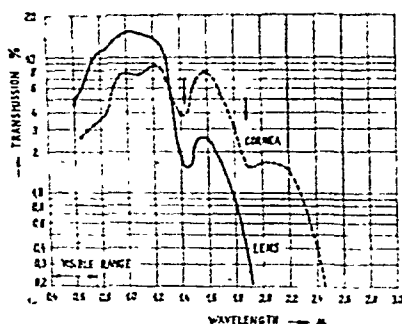


Fig. 1. Transmission characteristics.

FIG. 1.

having been removed during a transplantation. The characteristic was measured on a double rocksalt monochromator shortly after the operation. Fig. 2 shows the same cornea before the operation in visible light and Fig. 3 in infra-red light. Various degrees of opacity of lenses and corneae resulted in non-uniform spectral transmission characteristics, but the transparence always increases as we move from the visible to the near infra-red region and measurements would indicate up to now a maximum of the transparence of opaque lenses in the region of wave-length $\lambda = 10,000 - 12,000 \text{ \AA}$. The arrows in the diagram indicate absorption bands of water.



FIG. 2.

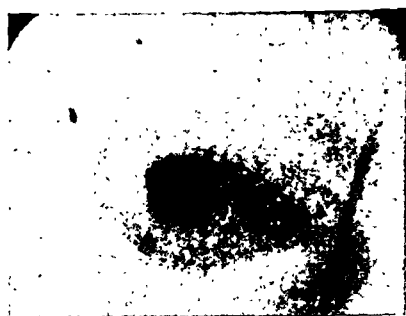


FIG. 3.

For observations of the anterior parts of an eye through an opaque cornea (*leucoma corneae post ulcus traumaticum*) the eye was illuminated with infra-red radiation of a wave-length exceeding $8,500 \text{ \AA}$ and by means of an objective lens an image was projected on to the photo-electric cathode of an image converter with a long-wave limit of $14,000 \text{ \AA}$. This image converter transforms the infra-red image of the observed eye on the cathode into a visible image on a fluorescent screen. This method of direct observation of the movable eye presents a number of advantages against photography on infra-red sensitive plates. We can simply focus by visual observation of the image on the fluorescent screen and photograph the image on the screen when the position of the eye is most suitable. Localisation in depth inside the eye can be estimated by employing parallax movement of the eye. Also we can use infra-red radiation of longer wavelength than can be used in direct photography, because the long exposures involved in photography on infra-red sensitive plates render this process impracticable for the living eye; therefore the infra-red

photographs which are known from literature all use merely the nearest parts of the infra-red spectrum.^{3 7}

The infra-red image converter which we used had also been adapted for use in conjunction with the customary apparatus for ophthalmological diagnosis (Nordensen slit-lamp).

Observation in infra-red light proved to be especially useful as a complement to the conventional methods of ophthalmological diagnosis before transplantations of the cornea and similar, nowadays frequently performed operations.

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"THE RETICULO-ENDOTHELIAL SYSTEM OF THE CORNEA" *†

BY

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THE results of investigations on colloidopectic properties of corneal cells are divergent. Whereas some writers as Blotevogel, 1924, Gasteiger, 1934, and others state that cells of the corneal stroma undoubtedly possess a colloidopectic ability, others as Loehlein, 1925, Jirman, 1936, did not succeed in confirming the observations of the above authors. Judging the question as one of far reaching theoretical and practical importance experiments were started on the permeability of cornea for some colloidal substances. Appropriate dye solutions and silver compounds were applied to the conjunctival sac. I attempted also to find those cells of the cornea which have the strongest properties of storing electronegative colloids, i.e., which cells of the cornea

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† From the Eye Clinic of M. Curie-Skodowska University. Director: Professor Dr. I. Abramowicz.

may show properties of elements of the reticulo-endothelial system.

Thirty white rabbits were used for the experiment. In 20 rabbits solutions of appropriate acid dyes, 1 per cent. trypan blue, izamin blue and carmin after Kiyon and alkaline dyes, 1 per cent. neutral red were dropped into the conjunctival sac during a period of 50 days.

The remaining 10 rabbits were treated with silver compounds 1 per cent. sol. arg. nitr., 5 per cent. protargol solution and 25 per cent. argyrol solution. Rabbits which had received acid and alkaline dyes revealed macroscopically a slightly discernible coloration. In microscopic preparations, however, no traces of dye deposits were found. In unstained sections 100-200 μ thick a rather uniform tint was stated which was undoubtedly the result of infiltration of all corneal layers with the applied dye which penetrates through the cornea into the anterior chamber similarly as through a half permeable membrane. Cells of the corneal stroma do not show colloidoptic abilities when colloidal solutions of acid and alkaline dyes are applied to the conjunctival sac.

That some corneal cells may reveal colloidoptic properties is proved by results of further experiments in which silver compounds were used. In contradiction to hitherto prevailing opinions it appeared that silver deposits in corneal argyrosis may



FIG. 1.

Corneal stroma. Stroma cells with numerous silver granules visible. Magnified $\times 550$.

occur almost exclusively in Descemet's membrane and that silver in form of deposits may occur in all corneal layers as well, except the epithelium. Silver deposits are partly located intracellularly within stroma cells, i.e., in sessile corneal cells partly outside the cells within structures of reticulin fibres in Bowman's and Descemet's membrane. Largest silver deposits occur within Descemet's membrane. These abundant deposits of silver granules within the elastic membrane are undoubtedly a consequence of some affinity of silver compounds to the so-called reticulin structures. Considerably smaller silver deposits in Bowman's membrane are most probably due to a partial resorption of silver granules by stroma cells. These transport the silver by means of plasmatic ways to the second reticulin membrane which constitutes a sort of a last barrier on which granules of silver compounds are held. This series of experiments proves that corneal stroma cells show distinct storing properties for silver, colloidopectic, and may be in some cases transformed into typical poliblasts, see micro-photograph.

We may therefore look upon corneal stroma cells as wandering cells in state of rest (Maksimow), which means that they correspond to prohistiocytes. Considering the above mentioned properties of stroma cells we may range them among elements which do not differ functionally from the fundamental components of the reticulo-endothelial system.

Summary

Colloidopectic abilities of the corneal stroma cells have been stated by means of a one per cent. Arg. Nitr. solution applied to the conjunctival sac of rabbits during a longer period of time. Stroma cells exhibit distinct properties in storing silver and are transformed in some cases into typical poliblasts. The opinion that stroma cells correspond to wandering cells in a state of rest seems to be justified. Corneal stroma cells may thus be ranged among elements of the reticulo-endothelial system.

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OPHTHALMIC CALCULATIONS BY THE "DAM" METHOD

BY

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If one should ask for example the average ophthalmologist what is the power of a contact lens having given radii of say 8.5 and 8 mm. and made of a given substance, say, $n=1.488$ the chances are he would be stumped even though he had once learned the method of procedure. Most likely the same reaction would be encountered if he were asked to calculate the power of the cornea or of an ordinary ophthalmic lens.

There is a method for calculating surface power and lens power which is really very simple and once learned can never be forgotten. I have called it the "Dam" method, and I don't mean "damn." It is based on the formulation of a unit of curvature comparable to the unit of power which is the dioptré.

Everybody knows that a 1.00 dioptré lens has a focal length or briefly a focus of one metre or 100 cm., or 1,000 mm., or approximately 40 inches. It is sufficiently accurate to take one metre as equal to 40 inches. The relation between power in dioptries and focal length, or briefly focus, is expressed by these "key" numbers. To get power from focus or focus from power we divide into one of these key numbers. For example, what is the focus of a 4.00 D. lens? Divide 4 into 100, gives 25 cm.; divide 4 into 1,000, gives 250 mm., divide 4 into 40 gives 10 inches. Or conversely, what is the power of a lens having a focus of 20 cm.? Divide 20 into 100, gives us 5.00 dioptries. If the focal length is given in inches, say, the focus is 20 inches, divide 20 into 40 which gives 2.00 D. and so on.

Exactly the same relation that exists between power (in dioptries) and focus in metres, centimetres, millimetres, or inches, exists between curvature and radius in the same units. The radius just like the focal length may be expressed in metres, centimetres, millimetres, or inches. The curvature is expressed in a unit of curvature called a metrec (term derived from metre-curve). The relation between curvature (in metrecs) and radius is expressed by exactly the same key numbers as used in the relation between power (in dioptries) and focal length. Thus what is the curvature of a surface having a radius of 20 cm.? Divide 20 into 100 and we get 5 metrecs. Or what is the curvature of a surface having a radius of 8 mm.? Divide 8 into 1,000 and we get 125 metrecs. Or what is the curvature of a surface having a radius of 10 inches? Divide 10 into 40 and we get 4 metrecs.

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The "Dam" formula is the general formula for surface power and is best written as $D=aM$. "D" stands for dioptries of power, "a" stands for the difference between the second index (that is the index of the medium where the light goes to) and the first index, *i.e.*, the index of the medium where the light comes from. It can be written as "a" equals N_2-N_1 . It may be memorised that "a" stands for the amount the second index is above the first index; M stands for the curvature in metrees. For example, what is the power of the anterior surface of the cornea having a radius of 8 mm. and an index of 1.376? The radius and index must be given or assumed. Using our "Dam" formula we get "a" = $1.376 - 1 = 0.376$, and $D = 0.376 \times \frac{1000}{8} = 47$ D.

Or what is the power of the *posterior* surface of the cornea given a radius of 6.8 mm. and the index of the aqueous as 1.336? Again $D=aM$, in which "a" is the difference between the second index, the aqueous humour on the other side of the surface, and the first index, which is that of the cornea itself (the posterior surface of the cornea separates the cornea from the aqueous humour). Here "a" equals $1.336 - 1.376 = -0.040$.

$$D = -0.040 \text{ times } \frac{1000}{6.8} = \frac{-40}{6.8} = -5.88 \text{ D.}$$

The " $D=aM$ " formula gives the power of a surface and combination of surfaces in terms of reduced power. This is the power used by Gullstrand and other writers on physiological optics. For a surface bounded by air, or for a lens as ordinarily used, *i.e.*, in air, the reduced power is the same as the actual power and we need not go any further into this.

In contact lens calculations since the radii are always given in millimetres the radius has to be divided into 1,000. If one takes "a" to three decimal places and then moves the decimal 3 places, which multiplies it by 1,000, one has only to divide this by the radius to get the power. Thus if a contact lens made of $n=1.488$ has radii of 8.7 and 8.5 mm. the surface powers will

$$\text{be } \frac{488}{8.7} = +56.09 \text{ for the anterior convex surface and } \frac{488}{-8.5} = -57.41$$

for the posterior concave surface. If the lens is infinitely thin the total power is the sum of the surface powers, in this case -1.32 D. In contact lenses the effect of thickness cannot be ignored and one must use a formula involving thickness.

This, however, is a secondary consideration and has to be used only after the surface powers are obtained. The "Dam"

method after being used for a couple of examples gives a simple and easily retained method for all lens problems. Say what is the power of a thin ophthalmic biconvex lens of radii 25 cm. and 40 cm., made of glass index being 1.52? Using $D=aM$ for

first surface, we get $D=.52$ times $\frac{100}{25} = \frac{52}{25} \approx 2.08$ D. For posterior surface figuring that surface is convex to the outside, and the first index is air, we get $D=.52 \times \frac{100}{40} = \frac{52}{40} = 1.30$ D.

Total power is +3.38 D.

The formula for the total power of a lens of any kind where the thickness has to be taken into account is not difficult to remember if one but thinks about it for a few minutes. The total or combined power is the sum of the surface powers with a modifying term. The formula consists of three terms, two of which are the surface powers and the third is the modifying term. Thus $D_p = D_1 + D_2 - D_1 D_2 e$. D_p stands for combined principal power, D_1 is the power of the first surface, D_2 is the power of the second surface. The sum of these two gives the basic power of the whole lens. The "modifying" third term is the product of these two powers, times the equivalent thickness "e." The equivalent thickness "e" is the actual thickness expressed in metres and divided by the index of refraction. Thus if the

thickness is 3 mm. and the index is 1.50, $e = \frac{0.003}{1.5}$, or if the thickness is .5 mm. and the index 1.488, "e" equals $\frac{0.0005}{1.488}$.

In short, the sum of the surface powers, minus their product times "e" gives the total power. The formula is best remembered when read just as written, "Dp equals D one plus D two minus D one D two e." In the contact lens example given above assuming the thickness to be 1 mm. the modifying third term is 56.09 times $-.5741 \times \frac{.001}{1.488}$. This works out to +2.16. Adding 2.16 to the sum of the surface power -1.32 gives us a true principal power of +0.84 D.

The effect of thickness in this instance is very marked. In most contact lenses it is very much less marked. In ophthalmic lenses in general the effect of the thickness is negligible. So that if one forgets everything except the "Dam" method he will still be able to calculate all surface power, all thin lens power, and at least approximately nearly all "thick" lens problems.

A NOTE ON THE POSITION OF THE EYE IN A THIRD NERVE PALSY*

BY

EUGENE WOLFF *and* Surgeon Commander HEFFERNAN

LONDON

It is generally stated that in a complete third nerve palsy the eye looks out and somewhat down or simply down and out due to overaction of the external rectus and superior oblique.

We would submit that this statement requires some qualification. The eye is certainly abducted but as in this position the superior oblique cannot depress the eye or only very slightly, the amount of depression is minimal or nil as in the following case:—

On November 18, 1946, G.W., aged 21 years, was involved in a collision while motor cycling.



FIG. 1.

* Received for publication, February 27, 1947.

He was taken to hospital and did not recover consciousness for five days.

When he came to, he noticed that he could not open his left eye. There was no other paralysis but he was told that he had had a temporary left sided hemiplegia for the first three days. He noticed that when he lifted the left upper lid with his hand he saw double. The diagnosis was fracture of base of skull, nasal bones and left jaw with left-sided third nerve paralysis.

When seen on January 20, 1947, we found as follows:—

Left eye closed, but he could lift the lid very slightly with the aid of the frontalis. Vision 6/6 in each eye.

Left pupil larger than right and fixed to light and convergence.

With the right eye looking straight forward and the left lid lifted manually (Fig. 1) the left eye was seen to be directed *directly outwards*. *The corneal reflexes were on the same horizontal level.*

On directing his right vision to the extreme right the left eye came to the mid-line. No other movement of the left eye was possible except in-torsion on asking the patient to look down. Both fundi were normal,

WAREN TAY-SACHS DISEASE IN A CHINESE INFANT*

BY

GOPAL HARIDAS

(FROM THE CIVIL GENERAL HOSPITAL, SINGAPORE)

A SURVEY of all the available literature on Tay-Sachs disease fails to reveal evidence of the record of a case previously from Malaya. I am thus submitting a full report of a case, which was admitted to the General Hospital, Singapore—the first case of its kind occurring in Malaya in a non-Hebrew infant—together with a brief review of the clinical types, history and symptomatology which I believe will not prove repetitious in spite of the extensive and controversial literature that has already been written on this subject.

Tay-Sachs disease is the infantile form of amaurotic family idiocy. Amaurotic family idiocy is a type of progressive cerebral degeneration which comes under the large group of the cerebro-retinal syndromes of the heredo-degenerative type. The following most modern classification of the clinical types of the cerebro-retinal syndromes of the heredo-degenerative type is given in the British Encyclopaedia of Medical Practice, Volume III:—

* Received for publication, March 29, 1947.

1. Tay-Sachs Disease (Infantile amaurotic idiocy).
2. Batten-Mayou Disease (Juvenile amaurotic idiocy).
3. Late Infantile Cerebro-Retinal Degeneration.
4. Late Juvenile Cerebro-Retinal Degeneration.
5. Anomalous Types.
 - (a) Tay-Sachs Disease without typical macular appearance.
 - (b) Batten-Mayou Disease with the cherry-red spots.
 - (c) Amaurotic idiocy without amaurosis.

Histological summary

In 1881, Waren Tay, an English ophthalmic surgeon, examined a 12-month-old male baby who was brought to him because he was unable to hold up his head and to move his limbs. At first, he made a provisional diagnosis of defective cerebral development and later examined the fundi. The examination revealed in the yellow spot in each eye in Tay's words "a conspicuous tolerably defined, large white patch more or less circular in outline and showing in its centre a brownish-red circular spot, contrasting strongly with the white patch surrounding it." Tay pointed out that this red spot was not due to haemorrhage or pigmentation but seemed to be due to thinning of the retina, thus forming a small hole or gap in the white patch which exposed the vascular choroid. When he examined the infant $4\frac{1}{2}$ months later, the macular appearances were unchanged but the optic discs which were first normal had become atrophic. The child had also become helpless and died when he was 20 months old. Subsequently three more male cases were observed by him in the same family and they died before the age of two years.

In 1887, Bernard Sachs of New York published a full report of a case of a peculiar form of idiocy associated with blindness. Later, a much younger sister of this patient also became similarly affected. The fresh brain at post-mortem was found to be unusually hard and the knife actually grated on removing a small section of the cortex. A histological examination of the cut cortex showed the protoplasm of the pyramidal cells to be in various stages of degeneration.

In 1894, Kingdon pointed out that Tay's description of the ocular changes and Sachs' description of the nervous manifestations both formed the important features of one and the same disease and in the same year Carter drew attention to the fact that the disease was confined to the Jews. In 1896, Sachs named the condition amaurotic family idiocy, and the name Tay-Sachs was suggested by Higier in 1901.

In 1909, Schaffer studied the histo-pathology of this disease:

in great detail. He characterised the minute pathology as an unusual swelling of the cell protoplasm and the dendrites. The interfibrillar material is first attacked and the cell degeneration follows. There results degeneration of every cell of the central grey matter of the brain, spinal cord and spinal ganglia and of the ganglion cells of the retina. There is attenuation of the nerve-fibre layer. The nerve cells later become filled with a granular pre-lipoid material. Schaffer concludes by expressing the opinion that children with amaurotic family idiocy possess "an abnormally exhaustive nerve-cell protoplasm, which, becoming paralysed with the strain of earliest functions, soon degenerates."

As the result of a few most important observations and elaborate microscopical examinations made by Poynton, Parsons and Gordon Holmes (1906), and by Mott (1907), the following conclusions were arrived at by them :—

(1) That amaurotic family idiocy is a primary disease of the nervous elements and that the proliferation of neuroglia is secondary to this.

(2) That it is a primary cell disease because the nerve-cells are relatively more affected than the fibres.

(3) That the primary change is in the interfibrillary part.

They also concluded that :—

(a) The disease was not due to arrested development because the symptoms were not present from birth.

(b) The disease was not due to bacterial toxins.

(c) The disease was due to some inherent biochemical changes in the protoplasm of the cell which led to its degeneration.

Recently histo-pathologists have shown that this disease is one of a group of primary metabolic disorders which leads to an accumulation of lipid substances, chiefly phosphatides in the nerve cells of the brain and ganglion cells of the retina.

Aetiology

Nothing definite is known of the cause of this affection. Syphilis, tuberculosis, alcoholism and neurotic taint play no part in its production. Infection has been excluded. It has been definitely established that it is a familial and racial disease. The occurrence of 111 cases in 69 families containing two or more members and the presence of consanguinity in the parents in more than 50 per cent. has brought out the recessive character of the condition. There is undoubtedly a greater incidence of this disease among the children of the Jewish race, especially the Jews of Poland and Russia, probably because consanguineous marriage is common

among this race. Sloane (1933) has traced 18 undoubtedly non-Jewish cases among the 200 or so reported in the literature. The case I am about to describe is a Chinese Hylam infant. A few cases have also been noted among the natives of Japan. Kamel Yacoub (1938) has reported five cases of Tay-Sachs disease, all in one Egyptian family.

Symptomatology of a typical case

The "clinical picture" of Tay-Sachs disease is so characteristic that when once seen it will never be forgotten. The infant is normal when born and develops normally up to the third or sixth month. He then becomes listless and apathetic and no longer seems to take notice of objects. As the weakness increases he is unable to sit up. The neck muscles are weak and his head falls backwards or forwards if unsupported. There is a progressive diminution of vision and the eyes wander aimlessly about. He is susceptible to sounds. There is optic atrophy and the cherry-red spot surrounded by a lighter halo at the macula. The limbs now become rigid, the arms are extended and rotated inwards at the shoulder, the legs are extended, adducted and crossed. The knee-jerks at this stage are usually exaggerated and the plantar reflex may give an extensor response. The cerebro-spinal fluid is normal. Wasting gradually becomes extreme. The hands become flexed and contracted. The feet become extended; the knee-jerks at this stage may be difficult to obtain. The infant lies in a semi-conscious condition, convulsions may occur and a squint may be present. If the infant is fed nasally he may be kept alive for some months in an emaciated condition. Death takes place from pneumonia or quite suddenly from cardiac failure.

Relation of Tay-Sachs disease to Niemann-Pick disease

The pathological changes in Tay-Sachs disease may serve as a basis of an opinion as to the relationship of this condition to Niemann-Pick disease. In 1928, R. Hamburger observed the occurrence of Tay-Sachs disease in a case of Niemann-Pick disease. In 1928, Bielschowsky made a histological study of a case and drew attention to this association of which a number of instances have been reported.

Niemann-Pick disease is a rare condition usually met with in Jewish children. Symptoms appear within a few weeks or months after birth. The spleen is considerably enlarged, the liver becomes enlarged and the infant becomes wasted and pale. Death occurs before the end of the second year.

Histological examination of the liver and spleen shows the presence of large "foam cells" distended with a yellowish lipid.

Similar cells are also found in the bone-marrow, suprarenal cortex and lymphatic glands, while the ganglion cells of the nervous system may be stuffed with lipoid. It is said that this lipoid in the ganglion cells does not represent the product of degeneration of the nerve-cell, but a faulty general metabolism with fat deposited in the nervous tissues. There is a striking similarity in the chemical composition of the abnormal cell contents in the liver in Niemann-Pick disease and of the brain in Tay-Sachs disease. Pick, Spielmeyer, Bielschowsky and Kufs have expressed the belief that Tay-Sachs disease has as its basis the same metabolic disturbance. However, in Tay-Sachs disease there is localisation of the general disturbance to the central nervous system alone. But Baker and Platou as the result of pathological findings in a case of amaurotic family idiocy they studied, are not inclined to agree with above view that Tay-Sachs disease and Niemann-Pick disease are related. They are inclined to agree with Schaffer (1935) that Tay-Sachs disease is an endogenous condition involving ectodermal elements and is not related to Niemann-Pick disease. Schaffer says that in Tay-Sachs disease there results a primary degenerative condition with swelling of nerve cells which later become filled with a granular pre-lipoid material and that Niemann-Pick disease was the result of a metabolic disorder. Von Bogaert's observation of cases of Tay-Sachs and of Niemann-Pick diseases, in the same family, is strong evidence in favour of similar pathology in these two diseases.

In addition to the symptoms already described, a large proportion of cases show mental degeneration, spastic paralysis, fits and blindness, in short the symptoms of amaurotic family idiocy and further the pathology of the nervous system in both diseases is identical. It seems probable that Niemann-Pick disease and amaurotic family idiocy are merely different clinical expressions of one and the same condition.

Report of the Case

Register No. : 464/40. Hoo See Ngee, a male Chinese Hylam infant, aged $17\frac{1}{2}$ months, was admitted to the Children's Ward, General Hospital, Singapore, on March 3, 1940, at 11.30 a.m. with a history of convulsions off and on for 4 months.

History : The infant was delivered normally at full term. History regarding consanguinity was not obtained because the parents never came back to see the infant after it was admitted into hospital. The infant is the fourth child in the family. The eldest child, a male, is healthy and his intelligence is normal. The other two children are dead and the cause of their death is not known. The infant was brought to hospital because he had been having fits for the last four months. He had been breast-fed for

three months after birth and then he was condensed-milk-fed. Rice water was given for some time. The child had been able to move his limbs, cry and recognise objects and mother up to four months before admission—that is until he was 13½ months old. He has never been able to sit up by himself but he could be made to sit up when supported by pillows. The mother said that the infant became less and less active as days passed on and up till four months ago she did not notice anything abnormal except that he was unable to sit up.

Examination.—He weighed 16 lbs. 14 ozs. on admission. He appeared pale and wasted. The circumference of his head was 46 cms. His head was large. He lay in bed listless and apathetic and took no notice of his surroundings. He did not follow a bright light and he took no notice of the feeding bottle. His eyes when opened were noticed to wander aimlessly about. His eyebrows were devoid of hair. He made occasional involuntary sucking movements with his lips. His mouth was kept open with everted lips.

Objects placed in his hand were not grasped. He could not take feeds from the bottle because he would not suck at the bottle. He would not swallow milk and medicines given with the spoon but they would only accumulate in the mouth. They had to be administered by means of the nasal catheter.

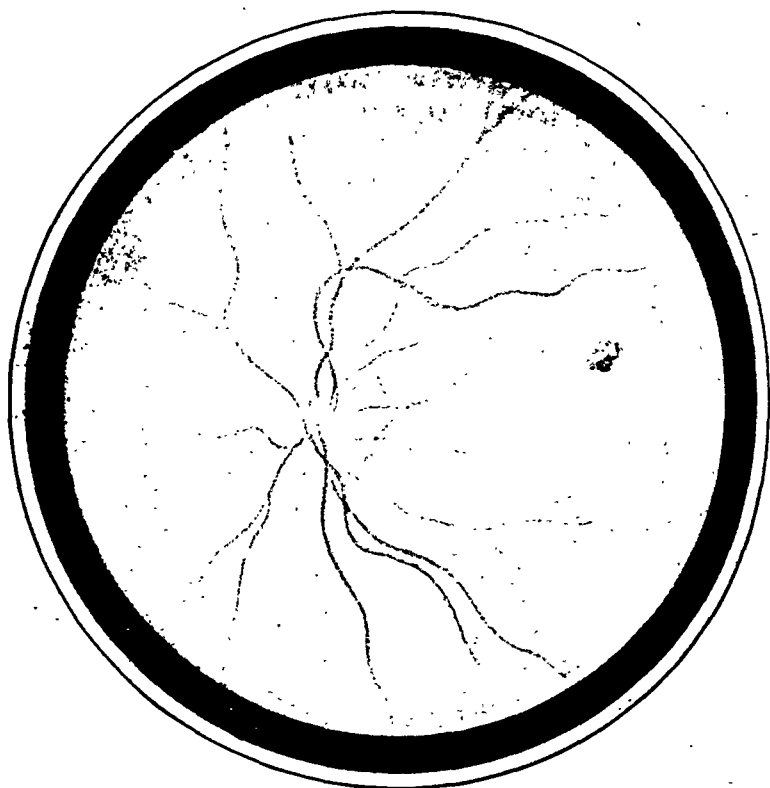
He lay in whatever position he had been made to lie without making the slightest attempt to move his limbs or turn on either side. The muscles were flaccid and toneless and the limbs were slightly rigid. One could occasionally notice some movements of the fingers of the right hand but the limbs as a whole were paralysed. The deep reflexes were exaggerated and the plantar reflex was flexor.

If he was made to sit up his head fell forward. He was sensitive to noises.

An examination of the fundus with the ophthalmoscope conducted under chloroform anaesthesia by Dr. A. D. Williamson, the ophthalmic physician and surgeon, General Hospital, revealed marked pallor of both discs, a somewhat oval dark purplish-red spot at macular areas (about $\frac{1}{4}$ size of disc) on a milky bluish-white background about three times area of disc.


An X-Ray examination of the skull, conducted by Dr. J. W. Winchester, Radiologist, General Hospital, Singapore, showed that the fontanelles were widely opened and that there was no direct evidence of tumour.

Progress: The infant used to have frequent attacks of convulsions. His head gradually became bigger—size of head on



Marked pallor of both discs; a somewhat oval dark purplish-red spot at macular areas (about $\frac{1}{4}$ size of disc) on a milky bluish-white background about three times area of disc.

March 3 was 46 cm. and on May 23 was 49.8 cm.—indication of a rapidly increasing hydrocephalus; on May 23 anterior fontanelle measurements

were $\begin{matrix} 7 \text{ cm.} \\ 5 \text{ cm.} \end{matrix}$  $\begin{matrix} 7 \text{ cm.} \\ 5 \text{ cm.} \end{matrix}$

On March 12 he had an attack of acute naso-pharyngitis and on March 15 he had enteritis. He got cured of these two conditions. On March 21 he developed a cellulitis of the scalp which gradually became worse.

Muscular wasting progressed to an extreme degree. The limbs became more rigid and all the deep reflexes were exaggerated. The arms were extended and rotated inwards at the shoulder, the legs were extended, adducted and crossed. The hands became flexed and contracted and the feet extended. There were numerous pressure sores all over the body. He had been kept alive by nasal feeding. He developed pneumonia and died of it on June 8, 1940,

that is when he was 20 months and 20 days old. An autopsy was performed but the autopsy notes are not available.

Laboratory examinations.—Blood cholesterol 186 mgm. per cent.; Blood Kahn Test Neg. (000); cerebro-spinal fluid Wassermann reaction Neg.; Cerebro-spinal fluid clear, not under pressure, nothing else abnormal.

Treatment: Antuitrins 1 c.c. daily intramuscularly for 6 days; and large doses of potassium salts.

Comments

This case of the infantile form of cerebro-macular degeneration is worth recording because it is the first case of its kind from Malaya in a Chinese infant. In the absence of a complete history, it is difficult to exclude the familial nature of the illness. Two other children in the same family have died, but it is not known of what they died. Although the age at which the first signs and symptoms manifested themselves is not clear and definite, the clinical features and course of the disease should be characteristic enough to make a clinical diagnosis possible but this was offset by the fact that this was a non-Hebraic case which confused the racial incidence and also by the fact that no such case had been seen before in Malaya. He was delivered normally at full term. He was admitted to hospital for convulsions when he was 13½ months old. But before this the mother had noticed that he had not been able to sit up by himself and that he had become less and less active as days passed on. After admission, it was noticed that there was marked muscular weakness and wasting, signs of visual failure ending in blindness and marked lack of mental development. An examination of the fundus revealed signs pathognomonic of the infantile form of cerebro-macular degeneration, a somewhat oval dark purplish-red spot at macular area on a milky bluish-white background. An additional feature in this case is a rapidly increasing hydrocephalus. He stayed in hospital for three months and five days and died when he was twenty months and twenty days old of terminal lobar pneumonia.

Summary

1. A case of the infantile form of cerebro-macular degeneration, otherwise known as Waren Tay-Sachs disease or amaurotic family idiocy, in a Chinese Hylam male infant is reported, the first of its kind from Malaya.
2. Due to an incomplete history, no history of consanguinity or of familial incidence was obtained.
3. The child was twenty months twenty days old when he died.

4. Diagnosis was established from the progressive muscular weakness, lack of mental development and blindness with pathognomonic changes in the fundus.

In conclusion, I have to thank Dr. William Heng for assisting me with the notes of this case, Dr. Chew Poh Kuo for the original diagram of the appearance of the left fundus, and the Director of Medical Services, Singapore Union, for permission to publish this case.

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TECHNICAL IMPROVEMENTS IN THE DIATHERMY OPERATION FOR DETACHMENT OF THE RETINA

BY

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THE diathermy operation for retinal detachment, in spite of its great development, still has such technical difficulties, which may be eliminated by improvement of the instruments used in the principal phase of the operation, the coagulative closing of the hole by the pointed perforating electrode.

There have been two main forms of perforating electrodes in use up to date: the needle, as described and used by Weve, Vogt, Szily-Machemer and others, and the nail of Safar and Arruga.

The drawback of the needle is that it has to be pulled out after every puncture. There arise leaking channels through the sclerotic and choroid from the beginning, with more or less continuous loss of inter-retinal fluid. Their number increases during the operation necessarily, hence the eye softens and the scleral surface

cannot be kept dry. Both circumstances make the further punctures more and more difficult, till at last it is sometimes impossible to continue with diathermic perforations, and one is obliged to change to other means, *e.g.*, to the cautery-loop.

Attempts have been made to avoid these inconveniences by shortening the working length of the needle and by refining its point. The depth of the puncture should not exceed 1.0—1.5 mm., so as to reach the choroid, but not to pierce it. Yet, this form had its own disadvantages, too. Too small an area of coagulation behind the retina could not always be seen by the ophthalmoscope, hence the control became doubtful. Moreover the coagulative effect would not suffice sometimes to arouse the necessary adhesive inflammation.

The nails, inserted, remain in place during the main phase of the operation. The Safar nail is still the best, yet set into the sclerotic its head protrudes 2 mm. above the scleral surface. Whenever the eye is turned back into the primary position—necessary for ophthalmoscopic control—the head of the nail is caught in Tenon's capsule. In consequence of this the head is either laid flat down on the sclerotic or lifted out. In the first case the point of the nail is vacillating inside, a great danger to the retina and the vitreous. Laid flat down on the choroid its discovery with the ophthalmoscope is rendered difficult. On the other hand, if taken out or loosened, it brings about the inconveniences of the filtrating channel. Besides it has eventually to be sought in the tissues. (Therefore nails should be as a rule always counted.)

There have been still more drawbacks in Safar's set. His insulated forceps did not seize the nail firm enough. During the insertion it readily tilts over. The forceps is too clumsy, its faces are too large, and sometimes it is difficult to get deep enough with it into a narrow slit.

All disadvantages can be eliminated by giving a better shape to the nail and by constructing suitable forceps for it.

This new nail is made of highly polished stainless steel. It has a round flat head, not thicker than 0.3 mm. and with a diameter of 15 mm. The length varies from 2 to 6 mm. It begins cylindrically and passes conically into a sharp point (Fig. 1).

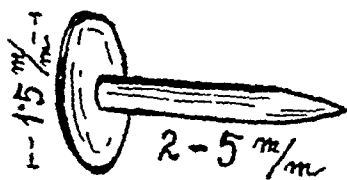


FIG. 1.

The new insulated forceps has angular ends. On the inner side of each end a small groove runs all along the three margins (Fig. 2). The parallel grooves work together and are designed to catch the nail.

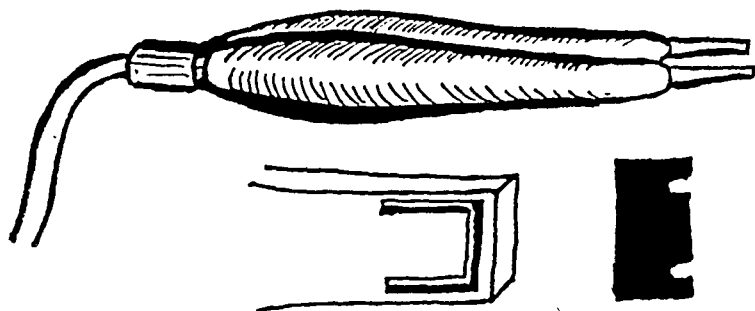


FIG. 2.

The isolation-forceps in full-shape, the grooved inner surface of the end and its cross-section.

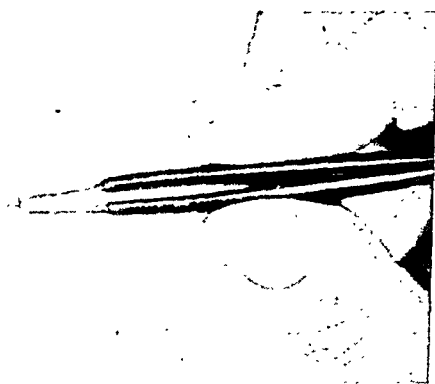


FIG. 3.

The nail seized with the isolation forceps perpendicularly.

The nail is easily seized by this forceps and directed as well in the axis of the forceps as perpendicularly to it (Figs. 3 and 4). The head of the grasped nail is held immobile in the forceps.

In order to keep the nails arranged at hand, and to protect them from being lost, we preserve them in cork-plates, taking them from there and putting them back there (Fig. 5).

Working with this equipment there are no disturbing moments in the course of the operation. As we use the nails not only for the closing of the hole, but at the same time for localisation, some



FIG. 4.
The nail seized vertically.

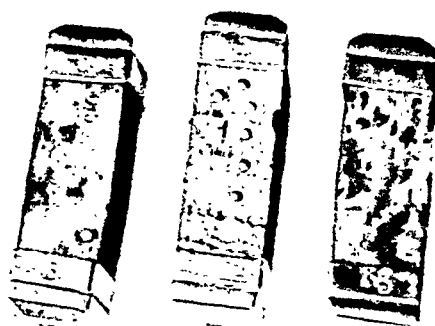


FIG. 5.
The nails arranged on cork plates.

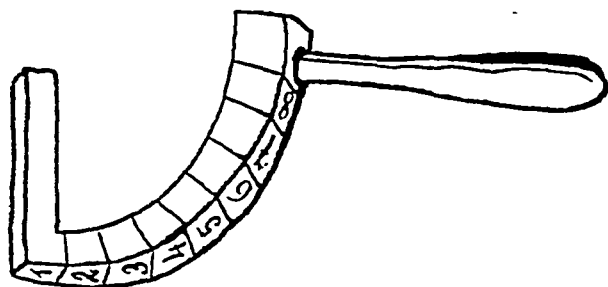


FIG. 6.
Small perimeter.

separate complicated method of localisation is superfluous, thus making the preparatory steps more simple. Briefly we proceed as follows:—

Meridian and limbus distance of the hole are determined by estimation and the perimeter. A small hand-perimeter (Fig. 6) allows direct ophthalmoscopy with simultaneous reading of the degrees. A plan is sketched in the known manner. We found an agreeable help an eyeball made of wood or paste-board. All necessary data are painted on its surface: cornea, ora serrata, insertions of the muscles, meridians and equator. The diameter of the ball is 44 mm., hence all measures divided by two are giving natural size. The hole is marked on its surface in pencil, thus we see the topographic relations directly and plastically.

We do not instil cocaine, thus diminishing the danger of the corneal surface becoming hazy. A drop of pantocaine suffices to be able to make the local anaesthesia with subconjunctival injections of novocaine.

The corresponding scleral surface is exposed and dried. The first nail is set in following the plan. The length of the nail depends on the diastasis of the retina in the region of the hole, yet it is advisable to choose a long one, say 5 or 6 mm., in order to find it quickly by ophthalmoscopy. With sufficient current (20—30 mA.) the nail plunges into the eye almost without resistance and the head adheres closely and immovably to the scleral surface.

With the ophthalmoscope the point of the nail is generally found immediately and its relation to the hole is established. With luck the nail strikes the right spot. Sometimes we see only the white spot of coagulation in the detached retina, corresponding to the needle behind it; that is sufficient for localization.

If the first nail is in a wrong position we settle the place of the next nail by estimation, *e.g.*, 3 mm. backwards meridianally and 2 mm. temporal or nasal longitudinally. The nail put in, new ophthalmoscopic control follows: It is superfluous to say that all nails set in remain preliminarily in their place. If necessary, we put in a third and fourth nail, till we did hit the hole or its margin somewhere, thus being able to get an easy start to put in all the other nails around the hole. These nails are shorter, 2—3 mm.

An example of an operation is shown in Fig. 7.

There is a typical hole of horseshoe form, meridian 2.30 o'clock, 13 mm. from the limbus. The first needle of 5 mm. hits the middle of the hole. With the following needles of 3 mm. length we mark the main points of the planned lines of coagulation, checking their

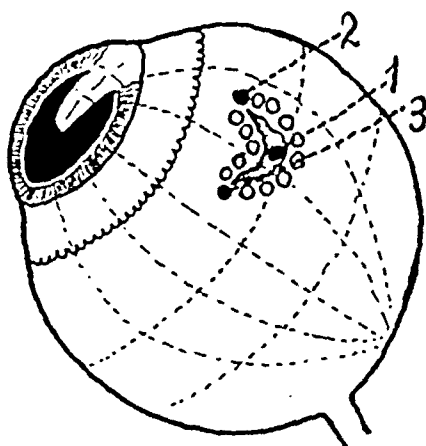


FIG. 7.

Plan and course of a detachment operation.

right position by ophthalmoscopic examination we fill up the lines with nails of 2 mm.

Having put in the last nail, an ophthalmoscopic control may follow, yet is not necessary.

The nails are quickly removed one after the other with the grooved forceps, and the operation field closed. (The nails are counted.)

There arises no harm from the nails in the wrong position or having perforated the retina. All such points give rise to small local inflammations, contributing to the adhesion of the re-attached retina.

It did not seem to be necessary to insulate the head of the nail or the upper part of its shaft, though it could be done. Bare nails compared with insulated needles did not show any difference in working.

For more than 3 years I have been using this set. Having gone through the whole evolution of the detachment-operation in the practice, I find it is superior to all other sets from the technical point of view. We make ophthalmoscopic examinations of the patient lying on the operating table and we start the operation only when we are certain of finding every detail of the fundus quickly and surely in this unaccustomed position. This and the technique together have contributed much to shorten the operation and to keep the cornea clear. The operation has become more exact, and this manifests itself in the increased percentage of the successful cases.

ANNOTATIONS

On the Decay in Proof Reading

It is odd that the more experience one has of reading proofs the worse one becomes at spotting typographical errors. We can only suppose that familiarity breeds contempt and that as one gets older one automatically pays less attention to what one reads. That we are not alone in this practice is evident in the perusal of an article in one of the daily newspapers of March 29, 1947, where the compositor has printed *habeus corpus* for *habeas corpus* and whoever read the proofs slipped up over the difference between "u" and "a." It is an easy mistake to make. But if one reads a book published in the early years of the last century one usually finds the proof reading to have been extremely good. Even with the old fashioned "long s," which is so closely similar to the letter "f," we did not notice a mistake in two volumes of about 500 pages each. Here, if anywhere, an error would be almost excusable. In the old days when the type was set by hand it should have been easy to pick out an "f" from the upper stratum of the case, and a "long s" from the lower stratum. And in using new type no error should be possible; but with old type which has been repeatedly in action it would be no wonder if, when the type was broken up, an occasional "long s" got into the "f" compartment and *vice versa*.

Maybe the having had to concentrate on type of this sort will have helped to improve our proof reading. It was quite curious to note how one would make mistakes over the "long s" and take the word "seed" for "feed." On rare occasions either would have made sense; but when we read what, at first sight, appeared to be "muffins" at the beginning of a line, we had mentally to scratch our head and look again. The word on closer inspection was "muslins." The paragraph dealt with the manufacture of commodities and however succulent a muffin of that era may have been it was obviously out of place in this connexion.

On Bindings

Our thoughts this month are devoted to bindings, by which we mean the covers of books not the binders that swathe the abdomen of the recently delivered female.

In olden times it was not unusual for the sheets to be sent out from the press bound in boards so that the purchaser could have the book bound in any style or material he liked. Plutocrats would choose a binding they liked and have the majority of the books in

their libraries bound alike. We have always thought that the results of this procedure were monotonous and would prefer to have different bindings for different sizes of books at the least. Some books are of much greater value uncut and in their original boards than in the most expensive form of binding.

A great deal depends on whether the owner wants his books to be read or merely looked at; some bindings, like some persons, wear very much better than others. School prizes used often to be bound in "tree calf," a form of binding that looks nice and shiny when new, but does not wear well. You do not need to be an authority on pig skins or law calf to settle the material for binding. Indeed, nowadays the question is settled for you by the publisher.

Provided a text-book be not too large and weighty, cloth forms a good binding. If a stouter cover is needed, buckram is excellent. Vellum, or half vellum, in any colour you like, gives a very good appearance and wears well. As to colour, this of course is infinite. The publisher will provide any colour in reason. We even remember having seen a book on colour blindness the cover of which was half red and half green. There is something to be said for a standard colour for professional text-books. Fuchs' first English translation came out in black cloth; the edition we bought in 1907 was in green, then came the red colour which has persisted till today. Nettlehip's little book was in green, Jessop's in blue, Henry Juler's in black and Sir George Berry's in green.

No one would wish to see the colour of a long set of volumes such as the *Transactions of the Ophthalmological Society* changed from its perennial green. Long may it continue! We always wondered who chose the colour of Vol. I. Was it Sir William Bowman, or the Council, the secretaries or the publishers? Search of the early minutes might answer the question, but like most of the utterances of Mr. Toots, it's of no consequence.

Much more important than the binding is the size and legibility of the print. It seems to us a poor economy to put a badly printed book into expensive covers for distribution as school prizes: but as these, we understand, are only to be looked at and never to be read it does not matter very much.

Their name liveth for evermore. *Ecclesiasticus*, xlii, 14

It is sometimes said that our profession is too fond of tacking the surname of anyone who makes a discovery on to it. The process can certainly be overdone, but there are some nominal appellations we could ill spare.

The circle of Willis, the Eustachian tube and the foramen of Monro are cases in point. And, may we, in parenthesis, remind

Southerners that the great Edinburgh anatomists did not spell their name phonetically?

Go where you will in medicine and its allied subjects you meet examples of what we are considering. The pharmacists have their Blaud's pill, Dover's powder and Easton's syrup among others. Our own specialty contains many examples and the names of Bowman, Meibomius, Argyll Robertson, Beer and von Graefe are always cropping up and their is hardly any telling where Hutchinson may not appear. We believe that Briggs was the first to describe the optic papilla, but if ever it had his name attached to it we have choked him off. Schlemm's canal is indispensable, and Herbert's pits save a deal of descriptive writing.

When all is said, we are no worse offenders than any other profession. A botanist who discovers a new flower, a sportsman who shoots a new antelope, an ornithologist who finds a rare bird, each is sure to have his name added to it as a memorial. And, for ourselves, we have always liked the principle. Hunter's canal must have been familiar to anatomists from very early times, but Hunter first made use of it to place a ligature on the femoral artery on the proximal side of a popliteal aneurysm and deserves an honourable tribute of this kind which will endure for all time. We see no reason why we should not continue to praise famous men in this way and the fathers that begat us.

BOOK NOTICE

The 1946 Year Book of the Eye, Ear, Nose and Throat. The Year Book Publishers Inc. Chicago, \$3.75. (Obtainable from Messrs. H. K. Lewis & Co., 136, Gower Street, London).

This new edition of the well-established Year Book of the Eye, Ear, Nose and Throat deals with the literature from August, 1945 to July 1946, and follows the lines of its predecessors. The contribution to ophthalmology occupies about 300 pages and is compiled by Dr. Louis Bothman of Chicago. The review of the year's literature is not, of course, comprehensive, but the author is to be congratulated on the choice of papers referred to as well as the readable way in which the subject matter is presented. The volume will be found useful to ophthalmic surgeons, but so far as practice in this country is concerned, it would be more so if it were divided into two with a complete separation of oto-rhino-laryngology from ophthalmology.

CORRESPONDENCE

THE "NORMAL" IN THE SYNOPTOPHORE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—I am glad to see that Mr. Emsley, in his letter in your last issue, confirms the very points that I, in my letter in your February issue, wished to make.

If I had said "qualities (as opposed to quantities) of heterophoria" I should perhaps have been more clear. It is precisely because it is not different "qualities" of heterophoria that the two instruments test, that the failure to check the zero on the synoptophore assumes importance when comparing the instruments.

We should, I think, all agree that the quantity of heterophoria revealed by any test depends upon the method of dissociation used. It depends also upon many other factors, some of which I have tried to examine in an earlier paper (*Brit. Jl. Ophthalm.*, p. 142, 1941) to which I would refer Mr. Emsley. My plea is for precision, where precision is possible, for an unchecked instrumental error obscures the very factors it is desired to measure.

Yours faithfully,

NIGEL CRIDLAND.

SOUTHSEA, HANTS.

May 22, 1947.

COLOUR VISION IN THE CONSULTING ROOM

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In the article by Dr. Neübert on "Colour Vision in the Consulting Room," May, 1947, p. 275. There are several points which require further explanation. The gross incidence of colour defectives is given in 5.5 per cent. which is considerably below the most recent estimates of the condition for the male population (7.8 per cent.). This low incidence may be due to the method of testing, no clear description of which is given. It is important to remember with the Ishihara plates that the light source must be good daylight or the test is of no value. With the Giles Archer Aviation Colour Perception Unit it is difficult to standardise the test, and no mention is made of the Test-Retest

reliability of this instrument on the same subject. This is a fundamental point where comparisons of different apparatus are to be made. In the later stages of the paper a testing lantern with three lights is quoted, but no mention is made of the constitution of this lantern. The only item of its construction which is given is that the source of light was regulated by a rheostat. Now such a method of control prevents adequate control of the light intensity and is also responsible for variations in the colour temperature of the source. Once again there is no reference to the test—retest reliability of the second type of lantern, and without that no useful comparison can be made. Finally, no attempt has been made to determine whether the second lantern is testing exactly the same thing as the first—colour contrast plays a part in the second lantern and not in the first, hence the two lanterns are not testing exactly the same thing.

In the absence of such information relating to the tests reported in this article, the author's conclusions about the relative sensitivity of the single light and the multilight lanterns are rendered invalid.

Yours faithfully,

JOHN GRIEVE.

MEDICAL SCHOOL, DUNDEE.

May 19, 1947.

NOTES

Excerpta Medica WE have received an announcement of this new publication together with the first issue of Section 13 (Dermatology and Venereology). The headquarters are in Holland and the Editor in chief is Dr. W. M. Woerdeman. The intention is to furnish a complete survey of all medical literature in the form of short abstracts. These abstracts are prepared by a staff of 3,000 specialists supervised by 400 editors. No details as to subscription rates appear in the announcement but these can be obtained from 111, Kalverstraat, Amsterdam, C.

* * * *

Appointments AT the last Council meeting of the Faculty of Ophthalmologists, Mr. J. H. Doggart was nominated the representative on the Editorial Board of the British Journal of Ophthalmology in place of Mr. Goulden.

H. B. Stallard, A. G. Cross and A. G. Leigh have been appointed Assistant Surgeons to the Moorfields, Westminster and Central Ophthalmic Hospital.

* * * *

The Illuminating
Engineering Society.
Annual General
Meeting.

At the Annual General Meeting of the Illuminating Engineering Society, on May 13, the President, Mr. J. S. Dow, presented the Report of the Council for the year ending December 31, 1946. Though recovery to normal conditions has not been as rapid as might be desired the Society has continued to make good progress both as regards activities and membership. For the first time the Council were able to report that membership has passed the 2,000 mark.

In the year under review a Centre has been opened holding new meetings alternately at Gloucester and Cheltenham.

The outstanding event of the year was the Society's first Convention, held in London, which proved a great success. The Society is now making arrangements for a Summer Meeting to be held at Harrogate from May 26 to 29, 1948, when the programme will include papers and social events. It is hoped that this initial meeting will be followed by other periodical gatherings in other areas.

The names of the following Officers for the next session were announced:—

President:—Dr. J. W. T. Walsh; *Vice-Presidents*:—Mr. J. M. Waldram, Mr. J. S. Preston and Dr. E. C. Walton; *Hon. Treasurer*:—Mr. J. C. Holmes; *Hon. Secretary*:—Mr. H. C. Weston; *Hon. Editor*:—Dr. S. English.

Dr. Walsh, the President-elect, is Chairman of the National Illumination Committee of Gt. Britain and is a Principal Scientific Officer in the Light Division of the National Physical Laboratory. He is well known to lighting engineers throughout the world as the author of several works on Illumination and Photometry which, though published over 20 years ago, are still regarded as standard works on the subject.

His election sets a new precedent, for this is the first time that any member of the Society has twice been elected to the Presidential Chair.

Finally the President announced the result of a ballot of members that had been taken on the proposal to form a Register of Lighting Engineers. Under the scheme the names of those corporate members who fulfil certain conditions may be included in a Register and are entitled to the exclusive use of the description "Registered Lighting Engineer (I.E.S.)." This proposal had been approved by a large majority.

On conclusion of formal business the President introduced Dr. N. A. Halbertsma, President of the International Commission on Illumination, who delivered an address entitled "International Relations in Illuminating Engineering." Dr. Halbertsma outlined the history and development of the Commission and explained the method of working of the Executive Committee and the National Committees of the various member countries. He also gave a number of amusing anecdotes to illustrate the difficulties met on account of the different languages spoken by delegates representing many countries at meetings of the Commission. Finally,

Dr. Halbertsma mentioned problems still before the Commission and mentioned that it was hoped that more countries will become members of the Commission in the future.

The next meeting of the International Commission on Illumination, the first since the outbreak of war in 1939, has been arranged to take place in Paris in September, 1948.

* * * *

Oxford Ophthalmological Congress **THE XXIIIrd Annual Meeting of the Oxford Ophthalmological Congress** will be held in Oxford, on July 3, 4, and 5, 1947. Mr. Williamson-Noble will be invested as Master on July 3, in the Department of Human Anatomy and will welcome the visitors. After the annual general meeting a discussion on "the contracted socket" will be opened by Professor Pomfret Kilner of Oxford and Mr. H. B. Stallard, of London. Mr. Tudor Thomas will read a paper on "Donor material for corneal grafts," and after an interval there will be papers by Mr. George Black, on "Some aspects of the treatment of simple detachment of the retina" and by Miss Savory, of London, on "Some uses of thrombin and fibrinogen in ophthalmic surgery." Tea will be taken in the gardens of Trinity College and the Annual dinner in Keble College Hall.

The second day opens with a paper by Mr. Currie, of Cheltenham, entitled "Eyesight and Industry." The Doyne Memorial Lecture will be delivered by Dr. Leon S. Stone of Yale University; it is entitled "Return of vision and functional polarization in retinæ of transplanted eyes." A paper by O. G. Morgan on "Some cases of inflammation of the other eye after cataract extraction" and a symposium of outstanding and instructive cases will be opened by Mr. Frank Law, Mr. H. Campbell Orr, and Mr. Philip Jameson-Evans. Tea will be taken in Keble College Gardens. On July 5 there are papers by Arnold Loewenstein on "Advances in anatomy and physiology obtained by clearing ocular tissues" and by J. P. F. Lloyd on "Making perimetry pay." A ciné film of "a ball and ring implant for use in enucleation," by Norman L. Cutter, of Wilmington, Delaware, is the last item on the programme.

* * * *

Third Pan-American Congress of Ophthalmology **DR. TOMAS R. YANES**, the President, and the Executive Committee of the Third Pan-American Congress of Ophthalmology have the honour to invite all ophthalmic surgeons to attend the event to be held in Havana, Cuba, from Sunday the 4th to Saturday the 10th, January, 1948.

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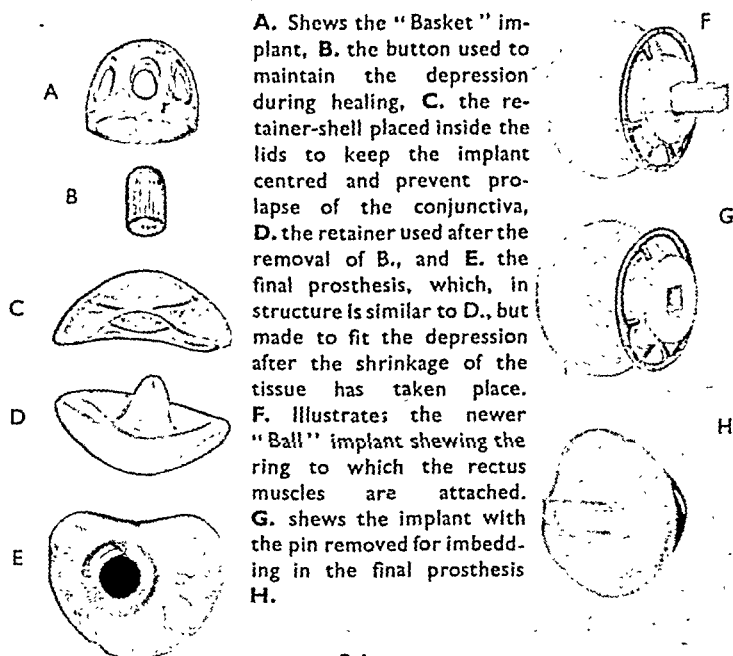
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CUTLER'S TWO IMPLANTATION SETS

The illustrations depict the two new plastic implants devised by Dr. Norman, L. Cutler of Willmington, Delaware, for use after enucleation to impart maximum movement to the prosthesis. Those on the left depict the original "Basket" implant and accessories, those on the right the later "Ball and Ring" implant, which provides a positive mechanical contact between the implant and the prosthesis.



A. Shews the "Basket" implant, B. the button used to maintain the depression during healing, C. the retainer-shell placed inside the lids to keep the implant centred and prevent prolapse of the conjunctiva, D. the retainer used after the removal of B., and E. the final prosthesis, which, in structure is similar to D., but made to fit the depression after the shrinkage of the tissue has taken place. F. Illustrates the newer "Ball" implant shewing the ring to which the rectus muscles are attached. G. shews the implant with the pin removed for imbedding in the final prosthesis H.

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THE BRITISH JOURNAL OF OPHTHALMOLOGY

AUGUST, 1947

COMMUNICATIONS

THE EFFECT OF ALUMINIUM AND ITS ALLOYS ON THE EYE :

A Report presented to the Vision Committee of
the Medical Research Council

BY

L. H. SAVIN

LONDON

Introductory

IN 1941 a young British sergeant was hit in both eyes by an exploding rifle grenade. One eye was disorganised and had to be removed. A piece of non-magnetisable metal from the grenade could be seen ophthalmoscopically in the vitreous of the second eye. Enquiry shewed that the metal was in all probability an aluminium zinc alloy. The patient was an intelligent man, desperately anxious for a prognosis. The following investigation arose from a desire to assist him.

Metallurgical considerations.—Jessop in 1915 gave details of the composition of the missiles then in common use. He found that bullets were made of lead with a mantle of steel or cupronickel. Shrapnel was composed of fragments of iron, lead, and mixed metal in a metal cylinder usually of iron. The common shell was

made of cast-iron or forged steel with a time-fuse of copper or aluminium. Hand grenades were of cast iron or glass. It will be seen that a high proportion of the missiles were of magnetisable metal.

During the first world war excellent papers were written on the technique of removal of magnetisable fragments by the electro-magnet, but little emerged on the prognosis and surgical treatment of non-magnetisable intra-ocular foreign bodies.

Between the two wars the use for industrial purposes of the aluminium alloys increased greatly. These owed their popularity to their cheapness, and the ease with which they could be adapted to any required purpose. They were light and non-magnetisable. Meanwhile came a marked development of non-magnetisable steels. In consequence the missiles of the recent war have been largely non-magnetisable. Stallard (1944) found that only 38 of 105 intra-ocular foreign bodies from his North African casualties were magnetisable; Savin (1943, also 1945) had an even lower incidence in his service cases—one out of every six, a ratio that obtained both at the beginning and toward the end of the war.

Pure aluminium is used commercially where maximum resistance to corrosion is required, *e.g.*, kitchen utensils and plant used in the chemical and food industries. Because of its good conductivity, the pure metal is also used for electrical conductors. Aluminium alloys contain relatively small amounts of alloying elements, copper, magnesium, manganese, nickel, silicon and zinc being most commonly used, either singly or, more frequently, in various combinations. The addition of alloying elements to aluminium improves its tensile strength and hardness, and aluminium alloys are therefore employed wherever high strength combined with light weight is required, as in aircraft production.

Depending on the manufacturing process for which they are best suited, aluminium alloys can be broadly divided into wrought and cast alloys; the former being produced and used in the form of sheet, strip, extruded sections, forgings, wire, etc., and the latter as castings. Another classification speaks of medium and high-strength alloys. Among the latter, duralumin, an aluminium alloy containing 4 per cent. copper, 0.5 per cent. magnesium and 0.5 per cent. manganese is the best known. In some of its mechanical properties duralumin is equal to mild steel. Another, even stronger alloy recently developed, contains 4–8 per cent. zinc, 4.0 per cent. magnesium and 3.0 per cent. copper as the main alloying elements. Nickel is added to improve the mechanical properties of aluminium alloys at elevated temperatures: thus an aluminium alloy containing 4 per cent. copper, 2 per cent. nickel and 1.5 per cent. magnesium (Y-alloy) is used for parts of internal combustion engines, such as pistons.

The more common cast alloys include the aluminium-10-13 per cent. silicon, the aluminium-copper-silicon, and the aluminium-10 per cent. magnesium alloys.

The alloying of aluminium, while it improves the mechanical properties, as a rule reduces the corrosion-resistance of the metal. This reduction in corrosion-resistance is especially marked in alloys containing copper and zinc, whereas the effect of magnesium, manganese and silicon is much less marked.

Since the war the Ministry of Aircraft Production has divided the aluminium alloys into standard groups with recognised variation of composition. The standards of these groups are laid down under specification for aircraft factories and foundries. The group numbers were not in use before the war; and are intended to minimise the mixing of various grades which was customary before that time.

1.—Review of the Literature

The literature of intra-ocular foreign bodies of aluminium is fragmentary and scattered.

The first references to such foreign bodies date to World War I (1914-18). In the 1913 edition of his monumental study of the injuries of the eye Wagenmann makes no reference to the subject; and the edition of 1926 dismisses the matter in a sentence: "During the world war intra-ocular foreign bodies of aluminium were frequently observed; they were generally minimal splinters; aluminium is relatively well tolerated, the inflammatory reaction being mild."

Handmann in 1915 and Pichler in 1918 drew attention to the appearance suggestive of synchysis scintillans following the entry of small foreign bodies into the vitreous. They recorded these foreign bodies as consisting of lead. Hertel in his review of ocular experiences during the 1914-18 war refers to these observations and states that he regarded such foreign bodies as consisting of aluminium, for unlike lead, they were not radio-opaque. A similar suggestion was made by Weigelin in 1917 in a study of non-ferrous intra-ocular foreign bodies; he regarded some ophthalmoscopically visible foreign bodies with a lustrous metallic sheen as "probably of aluminium," especially when they were negative to the sideroscope.

Whatever may have been the validity at the time of this distinction modern radiological technique has robbed it of value. The first clearly recognised case of an intra-ocular foreign body of aluminium was noted by Jess in 1924. Some workers on an underground construction lost a blasting cartridge made of pure aluminium without the addition of lead. It exploded on being

picked up in the street by a man aged 40 years. He sustained injuries to the left eye. There was a perforating wound 1-2 mm. long in the centre of the cornea, and a grey-white foreign body was embedded in the iris down and out with the point free in the anterior chamber. The corneal wound shewed numerous fine metallic dots giving the wound of entry the appearance of a silvery track. Fine silvery dots were also seen on Descemet's membrane near the wound and over most of the iris, especially towards the limbus. The foreign body did not respond to the sideroscope nor to the giant magnet, and X-ray did not shew any definite shadow. No attempt was made to remove the fragments, "owing to the repeatedly noted tolerance of the inner eye to aluminium." The eye rapidly settled down; and during the course of two years over which it was observed, the only changes seen were depigmentation of the pupillary margin near the foreign body, and some pigment deposition at the root of the iris. A faint lens opacity noted at the time of the accident did not increase in intensity.

In reporting his case Jess stresses that in his patient it was clearly known that the foreign body was of aluminium, while previous records had assumed foreign bodies were of aluminium on inconclusive evidence, or had dealt with non-magnetic intra-ocular foreign bodies in general.

The second case was reported by Fricke in 1925. A shooting instructor, aged 47 years, sustained injuries to both eyes from an exploding detonator, the casing of which consisted of aluminium. In the right eye there was a small perforating corneal wound. The surrounding corneal tissue, Descemet's membrane, the iris, and the anterior capsule all shewed numerous fragments of glistening silver-like metallic dust. Opposite the corneal wound, but considerably larger than it, there was a hole in the iris. The lens, vitreous and fundus were normal. There was no inflammatory reaction and recovery was good. The left eye suffered more severely. There was a large perforating wound at the limbus with prolapse of iris, turbidity of the aqueous, keratic precipitates, and restriction of visual field upwards. There was also fine metallic dust, distributed as in the right eye. The lens was opaque. Fricke holds that the aluminium did not affect the course of the recovery. The iridocyclitis of the left eye cleared up under treatment, and he ascribes the repeated recurrences as probably due to the associated apical phthisis from which the patient suffered.

Hesky in 1933 also described a case of aluminium in the anterior chamber. A mechanic, aged 42 years, came with a history that some five years previously he had been hit in the eye by a frag-

ment of pure aluminium. There was acute pain in the eye, and some blurring of vision. An X-ray as well as an attempted magnet extraction were apparently negative. The eye quieted down quickly and remained quiet for three years, when it suddenly became violently inflamed with deterioration of vision. Hesky noted circumcorneal injection and a corneal opacity covering a foreign body in the anterior chamber, the foreign body being visible through clear cornea when the patient was made to look sideways. X-ray examination had not revealed it. Operative removal of the fragment led to the clearing of the corneal opacity; and the lens, vitreous, and fundus were found normal. Examination confirmed that the splinter removed from the anterior chamber was indeed aluminium.

In addition to these clinical observations the following experimental records have to be noted:—

1. Jess in stressing the innocuousness of aluminium in his case reports mentions that he satisfied himself on this point by animal experiments. No further details are given.

2. Fontana (1938) reports three series of observations, each carried out on four rabbits:—

- (a) Implantation of splinters of aluminium in the anterior chamber. There were no inflammatory reactions. The eyes were enucleated after 55-85 days. Histologically the only change observed was some thickening of the corneal epithelium at the site of the incision scar.

- (b) Implantation of filings of aluminium into the anterior chamber. There were mild inflammatory reactions in the anterior segment of the eyes. The eyes were removed after 10 days. Histologically numerous particles of aluminium and some lymphocytes were seen in the corneal parenchyma and there was some atrophy of the ciliary body. The lens was clear.

- (c) Implantation of splinters of aluminium in the vitreous. There was no reaction and the eyes were enucleated after 30-64 days. Histologically there was mild lymphocytosis at isolated areas in the cornea and ciliary body.

In contrast to these relatively negative findings, Fontana reports three parallel observations on bronze. In each case severe destructive processes occurred.

3. Mielke (1941) briefly refers to animal experiments conducted over a period of two years. Pure aluminium and various aluminium alloys were implanted at ophthalmoscopically visible sites in the animal eye. Pure aluminium itself and some alloys were inert, whilst some other alloys liberated after a short while

flakes and corpuscles of metallic appearance. These could be seen floating freely in the vitreous. Mielke stresses that the inert behaviour of aluminium was unexpected in view of the purulent reaction aluminium splinters produce in the skin. As a practical corollary he advises against the "dangerous procedures" aiming at the operative removal of aluminium splinters from the interior of the eye. (The author refers to a fuller account of his findings in the *Ber. f. deut. ophthal. Gesellsch.* for 1940. This publication does not appear to be available at the present.)

In discussing the mild reaction of the eye to aluminium Jess held that this was to be explained by the "porous" nature of the metal. He believed that after penetrating the cornea an aluminium splinter broke down into powder. Fricke found this suggestion improbable in view of the hardness of aluminium. He would on the contrary expect a splinter of aluminium to retain its shape even if it were made semi-liquid by the heat of an explosion. In support of his views he adduces arguments bearing on the melting point and specific heat of aluminium in contrast to those of copper and iron.

A careful reading of the three clinical cases reported in detail hardly substantiates the view often expressed that aluminium is innocuous to the eye. In Fricke's patient there were recurrent attacks of cyclitis and Hesky's patient showed distinct if delayed intolerance to the aluminium fragment. There do not appear to be any cases recorded in detail on the effect of aluminium in the eye at a deeper level than the anterior chamber and there is therefore no actual knowledge of the effect of aluminium on the lens, vitreous or fundus.

Jess' and Mielke's experimental work is not reported in sufficient detail to allow adequate discussion. Fontana's experimental work with aluminum is given rather as a contrast to the violence of reaction of bronze, than as a considered study of the effects of aluminium. The experiments would have been more conclusive had the metal been left for longer periods in the eyes before enucleation. Even so, aluminium was not found by him to be completely inert.

The available data in the literature therefore suggests that an intra-ocular aluminium foreign body does not produce the destructive changes seen with iron and copper; but there is no reason to believe that the metal is completely innocuous to the eye. The stress hitherto laid on the inertness of aluminium by many writers seems to be made in a comparative sense to the severity of action of the heavy metals, rather than as an absolute statement based on a separate consideration of the effects of aluminium.

2.—Clinical Case Reports on Eyes with Intra-ocular Fragments of the Aluminium Alloys

The fluctuating circumstances of the war rarely allowed observation of patients for long periods. When the first war casualties began to arrive it was soon found that a high proportion of eyes were injured by fragments of aluminium alloy. Among the Dunkirk wounded several cases were seen where such fragments were embedded in the eye. One Algerian soldier had many small fragments of white metal dotted about in the vitreous. Afterwards other such cases were seen; but only two were watched long enough to provide adequate information as to prognosis.

CASE 1. On December 4, 1941, a young sergeant-instructor was injured by the premature explosion of a rifle grenade, made of "aluminium-zinc alloy."* He had been hit in both eyes. The left globe was disorganised and there were severe lacerations of both lids. There was a puncture wound of the right eye and a small piece of white metal could be seen in the vitreous. Several teeth were broken. His right tympanic membrane was ruptured. He had multiple puncture wounds of the right arm, chest, side of neck, and lips. There were injuries to the right musculocutaneous, musculospiral, and circumflex nerves. The left eye was removed on December 8, 1941. The right eye was carefully watched over a long period.

At first the piece of metal presented a bright and silvery appearance as it lay on the retina overlying the inferior temporal branch of the arteria centralis retinae about 6 disc-diameters from the papilla. Gradually the metal assumed a powdery white coating, and it could be seen that the retina in its neighbourhood was turning greyish in colour. Later on the metal was found to have shifted slightly leaving a mark or imprint on the retina to shew its original position. (Fig. 1.) On the most peripheral corner of the imprint there was a white incrustation which seemed to have separated from the white encrusted piece of metal, for the loose fragment now shewed a silvery exposed surface at one side. Later the fragment settled on the retina about two disc-diameters distant from its original position. Here it stayed for some months.

Later still the fragment shifted about $1\frac{1}{2}$ disc-diameters from its second seat of lodgment, and in its third resting place it finally disintegrated into a mass of powdery white debris. On January 6, 1944, the two retinal imprints could be plainly seen. (Fig. 2.) There was some retinal pigmentation in the neighbourhood of

* This was the description of the metal given by official sources. Mr. Freeman Horn, of the British Aluminium Company, Ltd., holds that the early corrosion and breaking up of the metal in this case would suggest that the fragment was a zinc-base aluminium alloy such as was commonly used for rifle grenades.

the white mass of debris near the second imprint. Since that date the appearances have remained unchanged. Meanwhile the patient has been boarded out from the service. The corrected vision in the eye is 6/5. He is earning his living as a clerk.

It can be judged that this particular fragment was not completely inert, as it disintegrated slowly and appeared to cause local patches of necrosis in the adjacent retina. There has been no evidence of any remote chemical action.

CASE-2. This patient was struck by a piece of German shell-casing, and there is only strong presumptive evidence, that the missile was in fact a piece of aluminium alloy. His Bren gun carrier was hit by a German 88 mm. shell on September 28, 1943, in Italy. He sustained burns on his back and left arm, and multiple puncture wounds of face, neck and arms. Both eyes were hit by shell fragments. Attempts to save the left eye failed. It was enucleated in North Africa on November 1, 1943, when it was found to contain several non-magnetisable metallic fragments.

He was admitted to Horton Hospital on December 10, 1943. The right eye was emmetropic with vision 6/18. The right cornea shewed a faint nebula with scattered patches of pigment thought to be of iritic origin on the posterior surface. There were patches of iris pigment on the anterior lens capsule. The lens itself seemed clear, as was the anterior chamber of the eye. There were many vitreous opacities, and two small pieces of metal were seen suspended in the anterior vitreous. These fragments were covered with a white coating and were very similar to the small oxide coated aluminium fragments which will be described later in the account of the rabbit experiments.

A large metallic fragment lay on the peripheral retina. Near it was an oval mark on the retina which was thought to be a local imprint as described in Case 1.

The patient was kept under observation for 7 months. During this period the vision dropped to 6/24. Vitreous opacities increased. The large metallic fragment gradually acquired a white coating, and the retina in the adjacent region gradually began to shew a diffuse pigmentation. For instance on January 5, 1944, the large fragment was clearly seen covered with a grey incrustation and surrounded by streaky retinal pigment which was also visible in streaks, dots, and lumpy aggregations in the adjacent retina. (Fig. 3.) The pigment appeared to be retinal rather than choroidal and was thought to indicate death of rods and cones in the pigmented areas. The "imprint" near the metal seemed to involve deeper structures: large choroidal

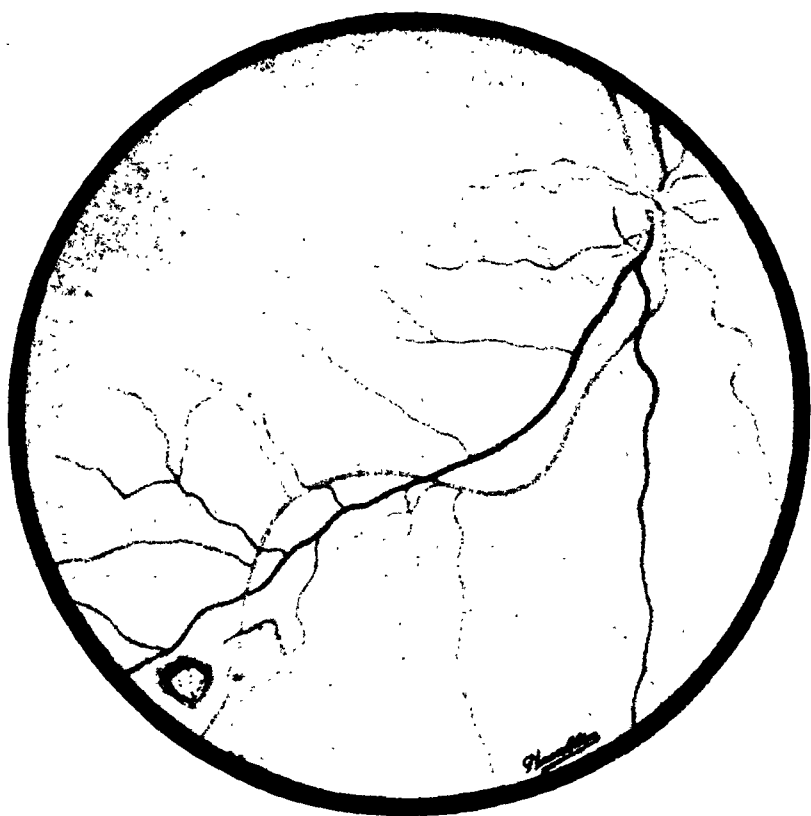


FIG. 1.

Sergeant W.J.H. Right fundus 11 months after entry of foreign body. The metallic fragment can be seen down and outwards, and the "imprint" indicating its previous site just above it.

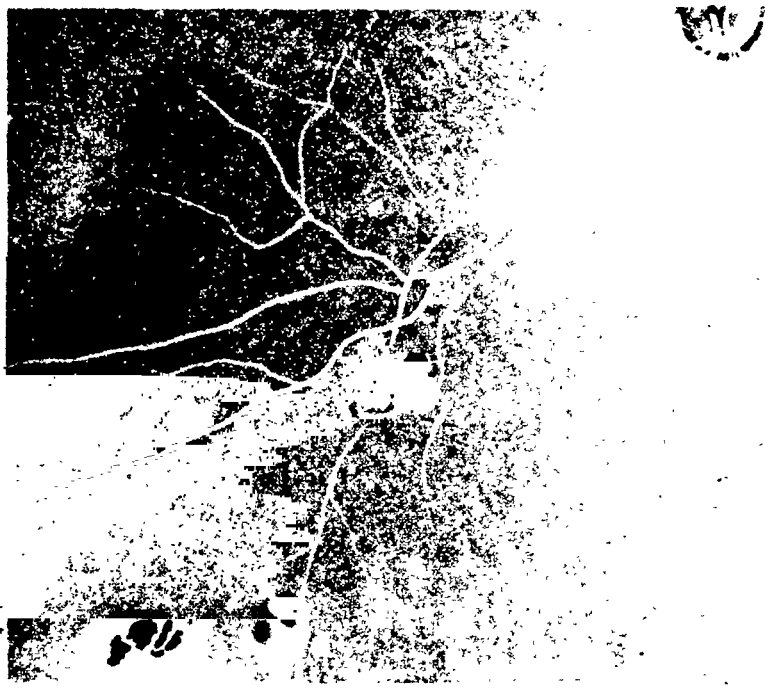


FIG. 2.

The same eye 25 months after the injury. The foreign body has shifted farther down, and two "imprints" are now seen. The fragment itself is breaking up. A mass of powdery white debris is present.

vessels could be seen traversing the patch. In the foreground could be seen a minute fragment of white-coated metal suspended in the vitreous.



FIG. 3.

Private R.N. Fundus appearance 16 months after injury. A large fragment is seen with a grey incrustation, and is surrounded by streaky retinal pigment in the adjacent retina. Below the fragment an "imprint" can be seen exposing the underlying choroidal vessels.

The patient has latterly been much troubled by swirling clouds of vitreous opacities. As there is still unabsorbed metal present in the eye, further deterioration in vision is likely. There seems no prospect of extracting the metal from this eye, the magnet having been tried unsuccessfully.

In this case besides local chemical reaction to the metal, there has been some more diffuse reaction, as shewn by the pigmentary changes on cornea, lens capsule, and in retina, and by the increasing vitreous opacities. It is unfortunate that no metallic fragments have been submitted to analysis.

3.—Experimental Observations on Rabbits

In preliminary experiments the animals were picked without any special regard to their fur colour. Presently it was realised that the pigmentation of a rabbit's fundus bears considerable relationship to its colour. In judgments of whether a fundus condition was pathological it was necessary to start with a standard type of fundus, excluding partial choroidal colobomata which were encountered unduly frequently in the rabbits available. Eventually a suitable standard rabbit type was selected from a silver-grey chinchilla-rex cross. This gave a light coloured fundus with minimal peripheral pigmentation. This was the type employed for the experiments with pure aluminium.

Only one animal of the series suffered from intercurrent disease; it was eliminated.

1.—STANDARDISATION OF TECHNIQUE

Standardisation was secured by the following measures:—

(a) *Anaesthesia*.—It was necessary to evolve a technique which would allow the implantation of metallic fragments in the rabbit's eyes without undue risk of death, without movement of the eyes under anaesthesia, and without post-anaesthetic complications. The eyes of the first three rabbits used were anaesthetised by instillations of 5 per cent. cocaine drops. In the first case it was thought that aqueous was lost rather rapidly from slight movements made by the animal. In the second case anaesthesia was satisfactory. The third animal started violently when the operation on its second eye was in progress causing a prolapse of iris and vitreous.

Subsequently general anaesthesia with a solution of nembutal gr. 1 to the c.c. given intravenously into the marginal vein of the ear was tried. About 0.75 c.c. was found necessary for a moderately large rabbit. At first smaller doses were used in conjunction with local cocaine anaesthesia. If the animal was too lightly anaesthetised, sneezing or reflex squealing sometimes occurred. Better results were obtained when slightly larger doses of nembutal were employed without cocaine locally. This ran rather near the danger-line. Two rabbits died almost immediately after receiving their injections apparently from sudden cardiac failure. A third rabbit seemed to recover satisfactorily and hopped round its cage. A few hours later it was found dead from no apparent cause. As it had been a healthy animal, the presumption was that death was due to some after-effect of the anaesthetic. Generally rabbits anaesthetised with nembutal recovered rapidly without any apparent after-effects from the drug. The possibility that the effects noted in the experiments might be changes caused

by a near-lethal dose of nembutal could be clearly eliminated, for such effects were observed in eyes which had been anaesthetised exclusively with cocaine, and were not observed in the unoperated eye of nembutal-anaesthetised animals which had aluminium or its alloys implanted in one eye only.

(b) *Technique for Anterior Chamber and Vitreous Implantations*.—To make the experiments strictly comparable a standardised operative technique had to be evolved. In proportion to the size of the globe the extra-ocular muscles in the rabbit are much thinner and flatter than in man. The nictitating membrane also has its muscles, and must be considered in planning operative procedures. Need of postoperative treatment or of dressings obviously had to be avoided. In the case of a vitreous implantation it was desirable to insert the metal so as to be easily visible ophthalmoscopically. The methods evolved proved their soundness in 26 successful implantations into the anterior chamber and 9 into the vitreous.

1. *Anterior chamber*.—Under anaesthesia as already described an incision 3 mm. long and 2 mm. distant from the limbus was made with a Taylor knife in the postero-superior corneal quadrant. The incision passed obliquely through the corneal stroma into the anterior chamber; and it was often possible to remove the knife without losing aqueous. The metallic fragment, sterilised by boiling in distilled water, was picked up by toothless curved iris forceps and inserted into the anterior chamber. An iris repositor was gently inserted to slide the fragment well into the anterior chamber. This manipulation sometimes had to be repeated several times where several fragments were inserted. Care was taken to select fragments of smooth surface to avoid unintended trauma.

This operation gave good results in general; but had the weak point that at the end there was little aqueous in the anterior chamber, and as the anterior chamber reformed the iris tended to adhere to the inner aperture of the corneal wound. An anterior synechia, usually only of thread-like consistency, occurred in 19 of the 31 operations. Filling the anterior chamber with saline might have lessened the incidence of synechiae, but was thought undesirable as introducing complicating factors, and rendering the technique less simple. In order to estimate the part played by operative trauma in the results, two eyes (rabbits 20 and 24) had the operation performed without the insertion of any fragment. Instruments were passed through the wound as in a standard implantation. Five days after the operation the wound in rabbit 20 had healed. There was a thread-like anterior synechia and a dot-like opacity in the lens at that site. This opacity was capsular and grew less marked

as time went on. In rabbit 24 there was also a small anterior synechia; but the eye quieted quickly and lens and media remained clear. In rabbit 19 an inert piece of metal (3 cms. of coiled-up platinum wire weight 15 mg.) was inserted into the anterior chamber by the usual technique. The entry wound healed quite uneventfully and without anterior synechiae.

2. *Vitreous chamber*.—Here the problem was to insert the metal through a puncture well protected from infection, and sufficiently far back to render the inserted fragment easily visible ophthalmoscopically; moreover vorticosc veins and other large vessels had to be avoided. A Graefe knife puncture was made behind the equator slightly posterior to the vertical meridian (*i.e.*, 10 o'clock for the right eye, 2 o'clock for the left eye); the blade was entered in a meridional direction, as delicately as possible, and not very deeply into the vitreous. In some instances there was a little bleeding, but less than would have been found after the equivalent operation on a human eye. Curved iris forceps proved the best instrument for the insertion of metallic fragments. It was rather common at first for the fragments to adhere to the forceps on withdrawal, this could be obviated by a slow withdrawal of the forceps.

There seemed few complications from this operation. Contrary to expectation no cases of detached retina occurred in the series. In rabbit 24 a control vitreous puncture was performed on one eye. The puncture healed rapidly and uneventfully.

No complications which might be construed as anything corresponding to sympathetic ophthalmitis in man nor any signs of infection were seen in the series.

Taking the experiments as a whole it was clear that operative trauma played a small, if any, part in the effects produced by the metallic fragments. It has been suggested that since the maximum life of a rabbit was approximately one-tenth that of a man, pathological processes might be correspondingly speeded up; no such acceleration was noted in these experiments.

2.—ANALYSIS OF EXPERIMENTAL FINDINGS

(a) *Changes in the metallic fragments*.—Most experiments were performed with 99.9 per cent. pure aluminium, which was employed in 16 eyes; in 2 eyes slightly less pure aluminium (98 per cent.) was employed. For purposes of comparison aluminium copper (group 2) was used in 4 eyes, aluminium magnesium (group 3) in 2, aluminium nickel copper (group 4) in 4, aluminium copper (group 6) in 2, aluminium silicon (group 8) in 2, and aluminium zinc (group 9) in 2 eyes. In one further case a piece of platinum wire was employed.

The only case where the metal was unaffected was that of the platinum. The 18 eyes tested with pure aluminium formed a homogeneous group. The various aluminium alloys were each tested in a few cases to see if there was much qualitative difference in their effect. It proved impossible to discriminate by the available clinical means between the behaviour of fragments of pure aluminium and fragments of alloy.

None of the aluminium or alloy fragments remained unaltered. Within a few days or weeks of insertion most fragments became coated with a thin film of powdery white material (9 instances). In some cases the film presented the appearance of a white or yellowish-white exudate and was thicker than the usual white coating (9 instances). In other cases there was a fibrinous coating to the metal (6 instances). This was confirmed in rabbit No. 3 which was killed 3 days after an anterior chamber insertion of aluminium copper (Group 2). The fragment was withdrawn in its coat from the anterior chamber and the coating of the fragment was investigated microscopically. A change, which on the whole developed rather later (10 instances) was the appearance of gelatinous material around the fragments—a development which sometimes ran parallel to absorption of the metal. A late effect observed in 11 instances was fragmentation or powdering of the metal; it was never seen before the sixth week. Some of the powdered metal was often observed in positions far from the original situation of the fragment. This may possibly have been connected with the lively movements and skittish behaviour of the younger rabbits. Sometimes these movements shifted the main fragments from one position to another inside the eye. Substantial or complete absorption of the metal occurred as a comparatively late change in 6 cases. The surface of such metal tended to assume a honeycomb appearance. Iridescence was sometimes observed to appear near a sharp edge or corner of metal, presumably indicating some chemical change. Table I shows the time sequence of these changes.

Changes in the fragments in relation to Dosage of Metal.—Doses employed ranged between 0.3 mg. to 20 mg. In the larger doses multiple fragments were usually employed. A dose of 0.3 mg. was employed in 8 instances, 0.4 mg. in 4, and 0.5 in 7. Doses of 0.9 mg., 4 mg., and 6 mg., were employed in 2 instances each, whilst doses of 1.5 mg., 3.32 mg., 4.98 mg., 6.64 mg., 8.0 mg., 8.3 mg., 10.0 mg., 12.0 mg., and 20.0 mg. were employed in single instances.

No alteration in the qualitative changes observed in the fragments appeared to occur when larger doses or multiple fragments were used. An odd feature was that when several fragments

TABLE I. *Approximate Time of Onset of Changes in Metal.*

(Pure aluminium O. Aluminium alloy X.)

Weeks	Coated white	Covered exudate	Fibrin	Jelly	Powdered	Absorbed
1	O	OOXX	O	-	-	-
2	X	-	XX	-	-	-
3	-	-	-	-	-	-
4	-	-	O	O	-	-
5	-	O	-	O	-	-
6	O	O	-	-	O	-
7	OX	X	-	X	ONX	-
8	X	X	-	-	X	OX
9	X	-	-	-	O	-
10	-	-	-	-	-	-
11	-	-	-	-	-	-
12	OX	O	-	-	XX	-
13	-	-	-	XX	-	-
14	-	-	XO	X	X	-
15	-	-	-	-	-	-
16	-	-	-	OO	O	O
17	-	-	-	-	-	-
18	-	-	-	-	-	-
19	-	-	-	-	-	-
20	-	-	-	-	-	OO
21	-	-	-	-	-	-
22	-	-	-	-	-	-
23	-	-	-	-	-	-
24	-	-	-	-	-	-
25	-	-	-	-	-	-
26	-	-	-	-	-	-
27	-	-	-	-	-	-
28	-	-	-	X	-	-
29	-	-	-	-	-	-
30	-	-	-	-	-	-
31	-	-	-	-	-	-
32	-	-	-	-	-	-
33	-	-	-	-	-	-
34	-	-	-	-	-	-
35	-	-	-	-	-	-
36	-	-	-	-	-	-
37	-	-	-	O	-	-
38	-	-	-	-	-	-
39	-	-	-	-	-	-
40	-	-	-	-	O	-
41	-	-	-	-	-	-
42	-	-	-	-	-	-
43	-	-	-	-	-	-
44	-	-	-	-	-	-
45	-	-	-	-	-	-
46	-	-	-	-	-	-
47	-	-	-	-	-	-
48	-	-	-	-	-	X

were employed in the same eye, while the alterations were similar in type, the various fragments shewed enormous variations in the degree they were affected. For instance one piece might be bright and shiny, while a second might be covered with a white powdery coat, or coated with gelatinous material. These differences did not seem to correspond to any constant factors. When multiple pieces were employed, they were cut from the same sheet of metal with the same stainless steel knife, and sterilised by boiling in the same test-tube. An impression was gained that changes were more rapid where two pieces of metal were in contact and at those edges of the metal which lay in actual contact with iris, lens, or cornea. As the multiple fragments were generally of the same shape, it was not thought that surface area was the factor involved; perhaps small differences in electrical potential play some part in these variations.

(b) *Local effects of fragments on adjacent ocular structures.* (Table II.) Fragments of aluminium or alloy which came into apposition with the internal ocular structures were frequently found to form a greyish necrotic patch in the subjacent structure. This "imprint" took the shape of the fragment, but varied slightly according to the situation in the eye. Sometimes in the iris and lens there was a tendency for the metallic fragment to sink into the soft tissues. In some cases the imprint became vascularised or occasionally pigmented. The incidence of imprints and their distribution are indicated in the last column of Table II.

Corneal imprints were noted in six eyes. In one case (rabbit 22, R. eye) the end of a fragment impinged on the posterior corneal surface. Three weeks later there was a diffuse haze in the cornea in this region and the end of the piece of metal was coated with jelly. Later the metal absorbed leaving a grey patch on the posterior corneal surface. Slit-lamp examination shewed metallic powder adhering to the patch. In a second case (rabbit 16, R. eye) a diffuse imprint formed round a piece of metal which gradually eroded forward into the corneal stroma. A third imprint (rabbit 16, L. eye) shewed as a pigmented plaque on the posterior corneal surface. A fourth imprint (rabbit 14, R. eye) shewed as an irregular diffuse patch containing a fragment of metal. A fifth imprint (rabbit 14, L. eye) was irregular in shape, and appeared to shew hyaline corneal degeneration. The sixth fragment (rabbit 2, R. eye) was observed at first coated with white and adherent to the posterior corneal surface. Eventually a vascularised scar formed containing metallic fragments.

Iris imprints were observed in 12 eyes. In one case (rabbit 4, L. eye) a grey patch was first noted when the piece of metal

TABLE II.—Ocular changes following *Intra-ocular Implantation of Aluminium and Aluminium Alloys. Aluminium.**

Anterior Chamber Implants

No.	Mg. Dose	Days Period	Cornea			Iris changes			Lens changes			Fundus changes			Imprint			Eye disorganised
			Nil	Slight	Gross	Nil	Slight	Gross	Nil	Slight	Gross	Nil	Slight	Gross	Cornea	Iris	Fundus	
2L	0.3	118	-	X	-	-	X	-	-	X	-	-	X	-	-	-	-	-
5R	0.5	207	-	X	-	-	X	-	-	-	X	-	-	X	-	X	-	-
5L	0.5	207	-	X	-	-	X	-	-	-	X	-	X	-	-	X	-	-
2R	1.5	118	-	X	-	-	-	-	-	X	-	-	X	-	-	-	-	-
13R	4.0	348	-	-	X	-	-	X	-	X	-	-	X	-	-	X	-	-
13L	4.0	348	-	-	X	-	-	X	-	X	-	-	-	X	-	X	-	X
14R	6.0	348	-	-	X	-	-	X	-	X	-	-	-	-	X	X	-	-
14L	6.0	348	-	X	-	-	X	-	-	X	-	-	X	-	X	X	-	-
16L	8.0	342	-	-	X	-	-	X	-	X	-	-	X	-	X	-	-	-
16R	10.0	342	-	-	X	-	-	X	-	X	-	-	X	-	X	X	-	X
22R	12.0	238	-	X	-	-	-	X	-	-	-	X	-	-	X	X	-	-

Vitreous Implants

8R	0.9	336	X	-	-	-	X	-	-	X	-	-	-	X	-	-	X	-
8L	0.9	336	X	-	-	-	X	-	-	-	-	-	-	-	-	-	X	-
17R	3.32	334	-	-	X	-	-	X	-	-	X	-	X	-	-	-	-	X
17L	4.98	334	-	-	-	-	-	X	-	-	-	-	X	-	-	-	-	-
18L	6.64	334	-	-	-	-	-	-	-	-	-	-	X	-	-	-	-	-
18R	8.3	334	-	X	-	-	-	X	-	X	-	-	X	-	-	-	X	-
23R	20.0	237	-	-	X	-	-	X	-	-	X	-	-	-	-	-	-	X

* The aluminium was 99.9% pure in all cases, except No. 2 in which metal, 98% pure, was used.

x = reaction present.

Aluminium Alloys

Anterior Chamber Implants

No.	Type of Alloy Group	Mg. Dose	Days. Period	Cornea		Iris changes		Lens changes		Fundus changes		Imprints		Eye disorganised
				Nil.	Slight	Gross	Nil.	Slight	Gross	Nil.	Slight	Gross	Cornea	
3R	2	0.3	3	X	-	-	X	-	-	-	-	-	-	-
3L	2	0.3	3	X	-	-	X	-	-	-	-	-	-	-
10R	4	0.3	323	-	X	-	-	X	-	-	X	-	-	-
10L	4	0.3	323	-	X	-	-	X	-	-	-	-	-	-
11R	6	0.3	235	-	X	-	-	X	-	-	-	X	-	-
11L	6	0.4	235	-	X	-	-	X	-	-	X	-	-	-
7R	8	0.4	336	-	X	-	-	X	-	-	-	-	-	-
7L	8	0.4	336	X	-	-	-	X	-	-	-	X	-	-
4R	9	0.4	215	-	X	-	-	-	X	-	X	-	-	-
12R	2	0.5	231	X	-	-	-	X	-	-	-	X	-	-
12L	2	0.5	231	X	-	-	-	X	-	-	X	-	-	-
11R	3	0.5	317	-	X	-	-	X	-	-	-	-	-	-
11L	3	0.5	317	-	X	-	-	X	-	-	-	-	-	-
4L	9	0.5	215	-	X	-	-	-	X	-	-	X	-	-

Vitreous Implants

9R	4	0.3	322	X	-	-	-	X	-	-	-	-	-	-	-	-	-
9L	4	0.3	322	X	-	-	-	-	X	-	-	-	-	-	-	-	X

shifted. Later the imprint was vascularised. The piece of metal in its new position sank slowly into the iris. In a second case (rabbit 5, L. eye) a white mass was first noted at the site of a partially absorbed piece of metal. A white iris imprint resulted. A similar white imprint was seen in a third eye (rabbit 5, R. eye). In a fourth case (rabbit 7, L. eye) the metallic fragment lay for some time on the iris surrounded with jelly. After the metal had become absorbed there was a grey iris imprint. In a fifth eye (rabbit 12, R. eye) a necrotic patch was noted on the iris after the metal shifted. In a sixth eye (rabbit 12, L. eye) there was a large grey imprint noted after shifting of the metal. A large area of iris atrophy resulted with new vessel formation in the adjacent iris. In a seventh eye (rabbit 14, R. eye) there were multiple grey iris imprints powdered with metal. In an eighth eye (rabbit 14, L. eye) a grey iris imprint gradually became depigmented and atrophic. A similar sequence of events occurred in a ninth eye (rabbit 13, R. eye). Three other eyes shewed grey iris imprints (rabbit 13, L. eye; 16, R. eye; 22, R. eye). Altogether vascularisation of an imprint was noted in 3 instances in the iris.

Lens imprints. The lens differed from other portions of the eye in being more susceptible to diffuse changes than to localised alterations. Lens opacities occurred in 28 instances; but these could be classified as "imprints" in 2 instances only. In the first (rabbit 1, R. eye) the lens capsule became opaque where a metallic fragment rested. Later a deeper opaque "imprint" formed in the lens. In the second case (rabbit 12, R. eye) a square fragment lay on the anterior lens capsule. The fragment shifted shewing a necrotic grey patch. In some instances pieces of metal resting on the surface sunk gradually into the substance of the lens.

Fundus imprints were noted in four instances. In the first (rabbit 8, R. eye) fundus examination shewed a metallic fragment far forward with an iridescent white coat. Behind it lay a pigmented scar thought to be a retinal imprint. Later the metal shifted. The scar was now seen to be dead-white with pigmented edges. In the new site of the metal the adjacent retina shewed pigmentation. In the second eye (rabbit 8, L. eye) a pigmented patch thought to be an imprint lay posterior to the metal. In the third eye (rabbit 18, R. eye) at one stage the metallic pieces were coated with a white film. Postero-superiorly was a white imprint, partially vascularised. In the fourth eye (rabbit 18, L. eye) near the lower end of a strip of metal a strand of gelatinous material extended from metal to retina. Powdery metal could be seen in the strand. Near this point were two white and necrotic imprint

areas. Later one of these patches shewed deep vessels (apparently choroidal) running across it, and pigmentation around it.

(c) *General Effects on the Eye.*—The general effect of the introduction of aluminium and alloy fragments into the eye, as opposed to the local effects already discussed, can be dismissed briefly. In view of the mobility of many of the fragments, a distinction between local and general effect cannot be maintained too rigidly, but it is convenient for descriptive purposes. An analysis of the effects noted is given in Table II.

The lens was one of the structures most commonly affected indirectly. Lens opacities were noted in 28 instances. The opacities varied in type, often in the same lens. Simple striae were noted in 13 instances; dots and vacuoles in 10, and irregular opacities in 10 instances. The opacities were mainly in the anterior capsule in 5 instances. In 6 cases the opacities were in the anterior cortex, and in 4 cases in the posterior cortex of the lens. In 4 cases polychromatic lustre was noted under the slit-lamp. As already noted there were two lens "imprints." In several cases fragments of metal sank into the substance of the lens. Oddities included two opacities in the form of a small ring. There were three complete cataracts of chalky white type.

None of the normal rabbits examined shewed any lens opacities. There seemed no doubt that the large number of opacities described were toxic in origin, either directly from immediate contact, or indirectly from disturbances in the nutrition of the lens arising from the action of the fragments on the uveal tract.

Inflammatory changes in the uveal tract were noted in a number of instances. These changes were usually gradual. Anything approaching the picture of an acute iridocyclitis with keratic precipitates as seen in man was noted in only two eyes. Posterior synechiae appeared in 10 instances, causing iris bombé in 2 eyes. Iris atrophy on transillumination was noted as a late change in 14 eyes. In one case the eye became shrunk and fibrosed.

Vitreous opacities were noted in 5 cases, blood in the vitreous in one.

The assessment of fundus appearances in the rabbits proved difficult, except in the case of gross changes such as the localised "imprints" already described in the neighbourhood of metallic fragments. A series of normal rabbits when carefully examined ophthalmoscopically shewed wide variations in the distribution and extent of their fundus pigmentation. The picture was complicated by some of the animals having pigmentation on the lower part of the fundus possibly connected with a tendency

to choroidal coloboma possessed by some of the strain. As already noted the degree of pigmentation bore some relation to the colour of the rabbits, and silver grey rabbits of special rex-chinchilla cross gave some approach to fundus standardisation. This standardisation was, however, approximate, as normal rabbits acquire more fundus pigmentation with increasing age. Of the 48 eyes experimented upon, 42 shewed initial patchy fundus pigmentation. In the course of experimentation 23 eyes developed pigmentation of what was thought a pathological degree, though this judgment was arbitrary. Six eyes developed obvious localised patches of choroiditis. Retinitis proliferans developed in three instances.

It must be concluded that toxic fundus changes were not uncommon, a finding confirmed by histological evidence.

A general reaction frequently observed was the formation of new vessels in the eye. For instance the corneal operation scar shewed superficial vascularisation in 15 instances; but in this situation the vessels could not be dissociated from direct operative trauma. In other parts of the eye this discrimination could be made. The cornea shewed superficial vessels in 10 instances. There was interstitial vascularisation in 2 eyes, deep vascularisation in two. New vessels formed at the limbus in 3 instances. Iris imprints became vascularised in 3 instances. In 2 eyes large vessels appeared on the lens capsule. Vessels appeared around metallic fragments in 3 cases. One fundus imprint became vascularised. New vessels appeared in the 3 cases of retinitis proliferans encountered.

Insufficient control experiments were performed to settle the question whether this common neo-vascularisation was in any way specific for aluminium or aluminium alloys.

A large number of isolated oddities were noted among the eyes used for experimentation. Two eyes developed bullous keratitis. One eye became buphthalmic (or staphylomatous) attaining a corneal diameter of 19 mm. There were two cases of zonular keratitis, one of interstitial, and one of deep keratitis. In one case cholesterol crystals appeared in the corneal stroma. Folds in Descemet's membrane were twice observed. In two cases pigment appeared on the corneal surface, once at the limbus.

(d) *Size and site of fragment.*—In the experiments employing alloys the total amount of metal introduced was 0.3 mg. in 7 cases, 0.4 mg. in 4 and 0.5 mg. in a further 5 cases. The difference in dosage was not sufficiently marked to enable any fine discrimination in the reaction of the eye to varying dosage of metal. None the less as can be seen from Table II, the suggestion emerged that 0.3 mg. was probably better tolerated than 0.5 mg. This is parti-

cularly seen in the case of fundus reactions. In the case of aluminium implants the dose varied considerably, ranging from 0.3 mg. to 20 mg., and here the correlation between dosage and effect emerges more clearly. Four eyes became disorganised; the dosage to which they had been submitted was 4 mg., 10 mg., 3.32 mg., 20.0 mg.; whilst the slighter effects on the cornea, iris, and lens generally tended to coincide with smaller doses, though exceptions were observed.

There was also some difference in the reaction of the eye according to the site of implantation. In the case of the alloys only two experiments in vitreous implantation were carried out, 0.3 mg. being inserted in each case. It is noteworthy that one of these two eyes became disorganised, constituting the only disorganised eye in the experiments with alloys. The second eye with vitreous implantation, though not ending in disorganisation, also ran a severe course. Implantations of alloys into the anterior chamber appear to have given the milder reaction. None of the 14 eyes ended in disorganisation, only two showed a gross corneal reaction, and one a gross iris reaction, whilst none gave a gross lens reaction. Gross fundus changes were, however, present in 6 of these cases. In the case of implants of pure aluminium 2 disorganised eyes were noted out of 11 anterior chamber implants, and 2 out of 7 vitreous implants. Comparison is difficult as neither the dosage nor the time factor were uniform. What is significant is that with the larger doses the site of implantation did not seem to matter much: all the tissues of the eyes appeared to be involved in the reaction. With smaller fragments the suggestion emerges that with anterior chamber implants the anterior segment of the eye was more prone to be involved than the posterior segment, whilst the opposite held true for vitreous implants.

That the site of the implant influences the position of the reaction is seen most clearly from the distribution of the imprints. The 6 corneal imprints, the 12 iris imprints, and the 2 lens imprints all occurred with anterior chamber implants, whilst the 4 fundus imprints were all seen with vitreous implants. Contiguity rather than dosage appears to be the determining factor as is seen in the case of the 12 iris imprints. Here the dosage of the implant ranged between 0.5 mg. to 12 mg.

4.—Illustrative Experiments

The following illustrative experiments bring out the reactions observed with pure aluminium and with the alloys. The ocular reactions to increasing doses of aluminium inserted into the anterior chamber and into the vitreous are illustrated by five and

six observations respectively, whilst single instances are given of approximately equal doses of five different alloys inserted into the anterior chamber and there is one instance of an alloy inserted into the vitreous.

A.—INSERTION OF PURE ALUMINIUM

(1) *Into the anterior chamber.*

(a) Effect of 0.5 mg. observed for 4 months.

Rabbit 5. A fragment of 0.5 mg. of 99.9 per cent. pure aluminium was inserted into the left anterior chamber. Four months later the appearance was as in Fig. 4. The corneal scar was firmly healed, but included a black patch of iris pigment. Near the scar the iris showed a grey irregular imprint at the former site of the fragment. The piece of metal had disappeared and could nowhere be seen.

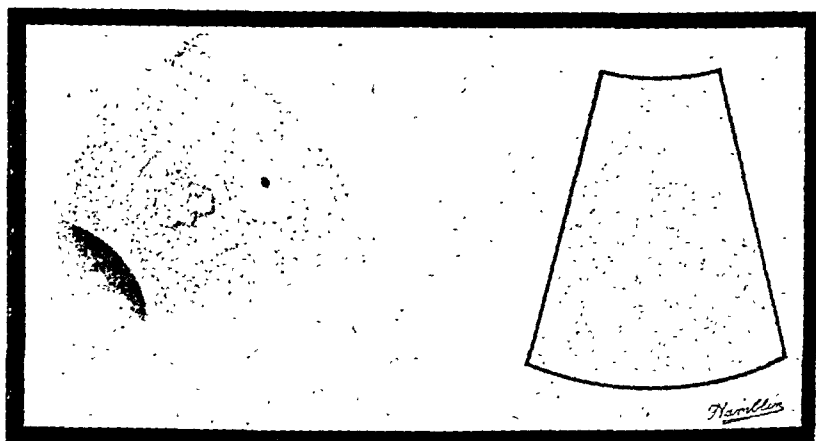


FIG. 4.

Rabbit No. 5. Appearance 4 months after implantation of 0.5 mg. pure aluminium into the anterior chamber.

(a) An "imprint" is seen on the iris. The metal itself has disappeared. Some iris pigment is visible in the incision scar.

(b) Pigmentary and necrotic changes are seen at the peripheral fundus:

The fundus showed peripheral pigmentary changes and some small and necrotic white patches. Many lens opacities were visible.

(b) Effect of 4 mg. observed for 10 months.

Rabbit 13. Through a postero-superior corneal incision 4 mg. of 99.9 per cent. pure aluminium were inserted in 1 mg. fragments into the anterior chamber. Soon the corneal scar became vascularised. Two of the pieces of aluminium sank into the lens; the other two fell to the lower angle of the anterior chamber and

became covered with jelly and incrustation. After a while posterior synechiae formed and were followed by iris bombé.

Ten months after insertion the appearances were as in Fig. 5. The lower third of the cornea presented a grey opacity in its deep

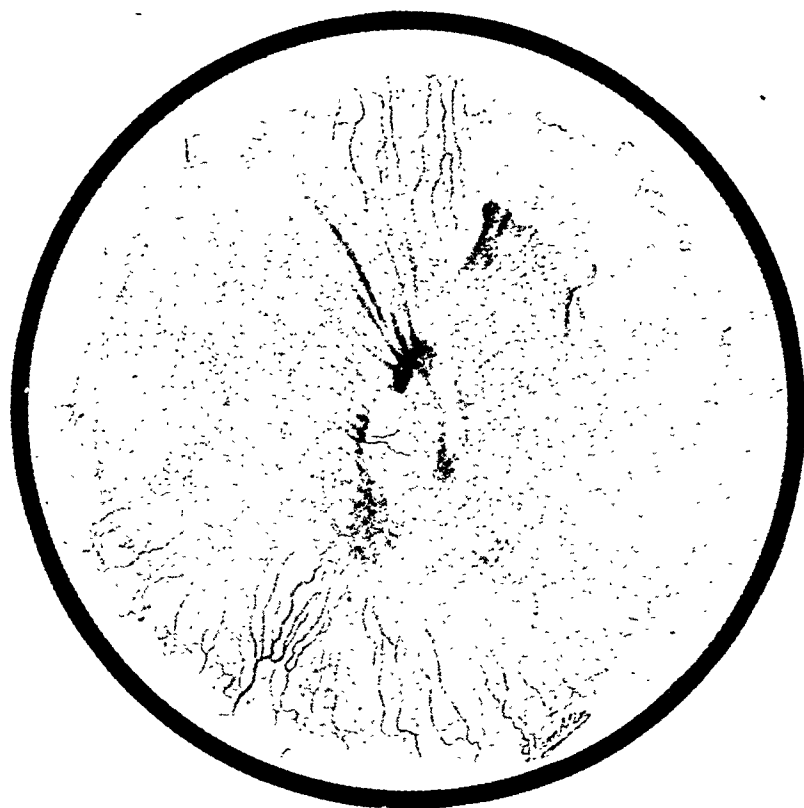


FIG. 5.

Rabbit No. 13. Implantation of 4.0 mg. pure aluminium into the anterior chamber. Appearances after 10 months. The lower third of the cornea shows a grey opacity in its deep stroma. Opposite this area a jelly, derived from the foreign body, has previously formed. The corneal opacity shows superficial vascularisation. The iris shows severe inflammatory changes with extensive posterior synechiae and the formation of iris bombé.

stroma. This was the area which had been opposite the jelly previously formed. This area of cornea presented much superficial vascularisation, the fine newly formed vessels taking origin from the limbal vessels. There was also much superficial marginal vascularisation at the limbus above.

The corneal scar shewed fine vascularisation; and there was a broad anterior synechia attached to this entry scar.

In the lower angle of the anterior chamber lay the partially absorbed remains of one of the fragments, coated with white jelly and exudate.

In the pupillary area was a nodule of lens matter. This had a few fine vessels on its surface, which appeared to be rising from the anterior surface of the iris. The pupil was firmly adherent to the lens capsule, and was blocked with fibrous tissue. Much of the iris, particularly below, shewed iris bombé.

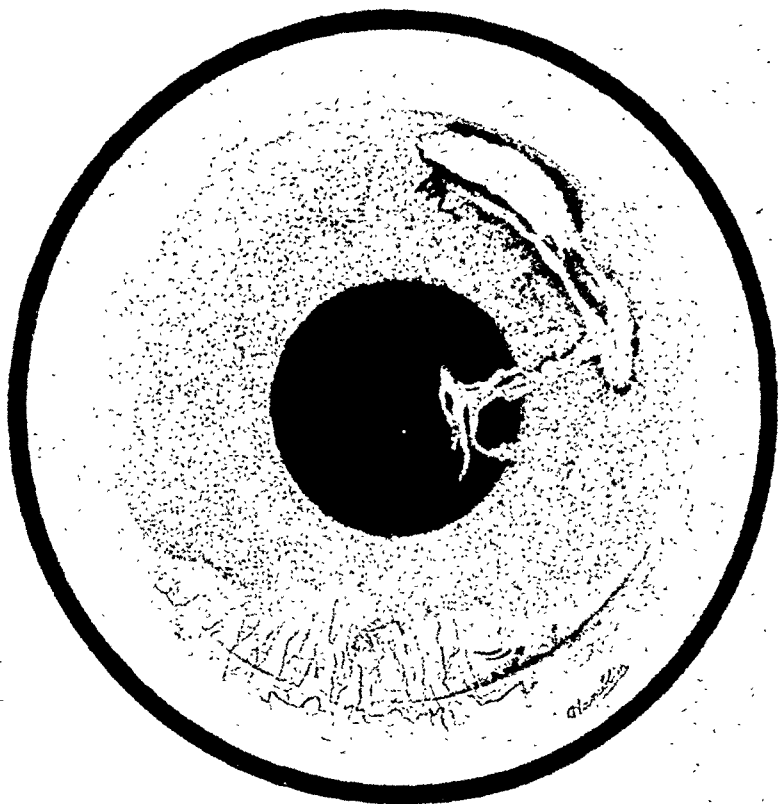


FIG. 6.

Rabbit No. 14. Implantation of 6.0 mg. pure aluminium into the anterior chamber. Effects seen at 10 months. The aluminium had been inserted in 6 fragments of 1 mg. each. There is diffuse opacification in the lower part of the cornea. Superficial vascularisation and increased limbal pigmentation are present. A corneal "imprint" is seen in the pupillary area. Transillumination showed the iris to be atrophic. Over the site of insertion of the metal a large grey iris imprint with pigmented margins is seen as shown in the upper part of the illustration.

(c) Effect of 6 mg. observed for 10 months.

Rabbit 14. Through a postero-superior corneal incision 6 mg. of 99.9 per cent. pure aluminium was inserted into the left anterior chamber in fragments of 1 mg. each. Ten months later the appearances were as in Fig. 6. The lower portion of cornea shewed an opacity of a diffuse nature in its deeper layers near the angle of the anterior chamber. This area of cornea shewed superficial vascularisation arising from the limbus and confined to the area of the opacity. Limbal pigmentation was increased and the pigmented patches encroached at some points on to the corneal epithelium. Opposite the pupillary area there was a posterior corneal imprint of irregular shape. The iris was atrophic as shewn on transillumination. Near the site of insertion of the metal was a large grey iris imprint with pigmented margins. The

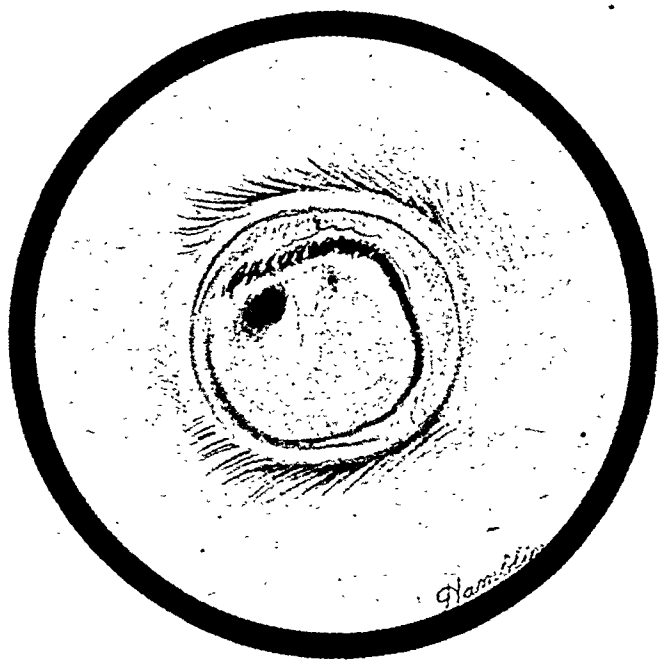


FIG. 7.

Rabbit No. 16. Implantation of 10 mg. pure aluminium into the anterior chamber of right eye. Changes seen after 10 months. The aluminium had been inserted in 10 pieces of 1 mg. each. After undergoing the milder changes seen in the earlier cases, including pathological fundus changes, the eye became enlarged simulating buphthalmos. The corneal diameters measured 19 mm. Diffuse oedema of the corneal epithelium and an interstitial keratitis with much vascularisation, are present. The conjunctiva is heavily vascularised.

lens shewed many dots and striae. In the lower angle of the anterior chamber some of the metallic fragments could be seen coated with exudate and jelly, and dotted here and there with pigment.

(d) Effect of 10 mg. and 8 mg. respectively observed for 10 months in the two eyes of the same animal.

Rabbit 16. Under nembutal anaesthesia 10 pieces each weighing 1 mg. of 99.9 per cent. pure aluminium were inserted into the right anterior chamber, and 8 similar pieces into the left.

In the right eye the metal underwent the usual changes of becoming coated with white material and jelly, vascularisation and absorption. Iris and corneal imprints formed. Some of the bits of metal sank into the iris stroma. Pathological fundus changes appeared. Ten months after implantation the eye presented the appearances shewn in Fig. 7. The eye was now diffusely enlarged and looking superficially like a case of buphthalmos, though closer inspection shewed that the detailed appearances differed and were rather those of a general staphyloma. The corneal diameter was 19 mm. There was a diffuse oedema of the corneal epithelium and an interstitial keratitis with much vascularisation. Through the cornea could be seen masses of gelatinous material and fragmented pieces of metal. There was much increase of pigmentation at the limbus. The conjunctiva was heavily vascularised. Transillumination shewed that the iris was atrophic and that there were anterior synechiae. The lens and fundus were not visible at this stage.

Meanwhile progress of the changes had been much slower in the left eye. Appearances ten months after insertion of fragments were as in Fig. 8. Here there was a large posterior corneal imprint opposite the pupillary area, with a diffuse pigmented plaque on the posterior corneal surface. Several pieces of metal covered with white exudate and jelly could be seen at the lower angle of the anterior chamber. Opposite the fragments the cornea was opaque on its deep surface and vascularised superficially. The metallic fragments shewed signs of partial absorption, and powdery metal could be seen on the anterior surface of the iris which was atrophic. The lens shewed streaky opacities. The left fundus shewed much diffuse pigmentation, which was considered pathological.

(2) *Into the vitreous.*

(a) Effect of 0.9 mg. with picture at 13 weeks.

Rabbit 8. Fragments of 99.9 per cent. pure aluminium weighing 0.9 mg. were inserted through scleral puncture into the vitreous. Thirteen weeks later the appearances were as in Fig. 9. The fragment lay on the peripheral retina covered with a greyish-white coat. The fundus generally shewed a large number of small



FIG. 8.

Left eye of the same rabbit. (Rabbit No. 16.) Here 8 strips of aluminium 1 mg. each had been inserted into the anterior chamber. The reaction, observed for the same period of 10 months, ran a milder course. A large corneal "imprint" was present in the pupillary area with a diffuse pigmented plaque on the posterior corneal surface. Several pieces of metal covered with white exudate and jelly can be seen at the lower angle of the anterior chamber. Opposite these fragments the cornea tends to be opaque on its deep surface and vascularised superficially. Some disintegration of the metal can be seen, as also white powdery debris on the anterior surface of the iris which is atrophic. Lens opacities are present.

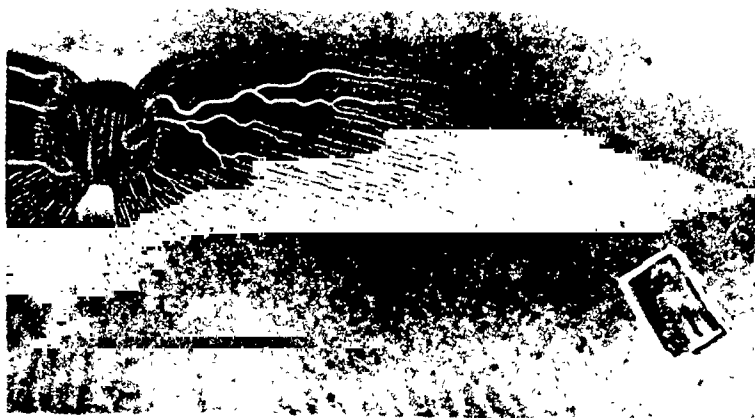


FIG. 9.

Rabbit No. 8. Implantation of 0.9 mg. pure aluminium into the vitreous. Changes observed after 13 weeks. The fragment is seen to lie peripherally on the retina, and shows a greyish white coat. A number of small necrotic white patches are seen throughout the fundus. The inset shows the striae which were seen in the lens of the other eye.



FIG. 13.

Rabbit 18, left eye. Insertion of one piece of aluminium weighing 6.64 mg. into the vitreous. Appearances observed at 7 months. Two large white imprints are seen with a smaller one at the end of the bent strip of metal. The metal itself is eroded and coated with white exudate. A strand of gelatinous material extends from the metal to the junction of the two main imprints. Part of the metal can be seen in this strand and scattered over the nearby fundus. There was pathological pigmentation in the periphery of the fundus.

necrotic white patches. The inset to the figure shews the striae which were seen in the lens of the other eye.

Later on the metal shifted leaving a retinal imprint. Pigmentation increased in the fundus.

(b) Effect of 3.32 mg. and 4.98 mg. with illustrations at 9 months and 7 months respectively in the two eyes of the same rabbit.

Rabbit 17. Four fragments of 99.9 per cent. pure aluminium each weighing 0.83 mg. were inserted into the right vitreous through an antero-superior scleral puncture behind the equator of the eye. There was much difficulty in making the fragments

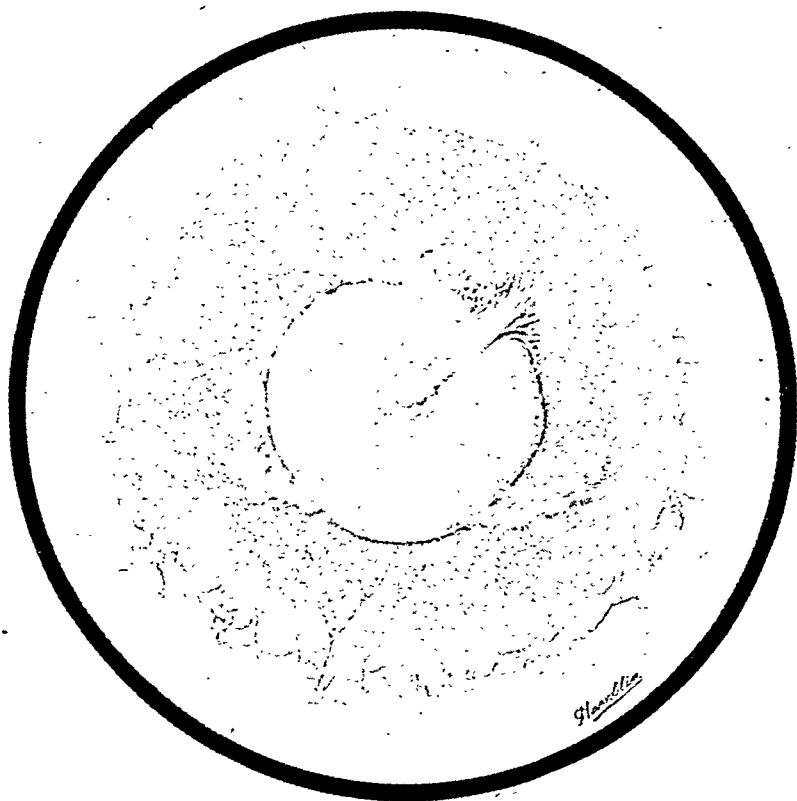


FIG. 10.

Rabbit No. 17, right eye. Implantation of 3.32 mg. of pure aluminium in 4 fragments into the vitreous. Changes observed after 9 months. The eye is shrinking; the corneal diameter measured 12 mm. as opposed to 14 mm. for its fellow. Marked superficial keratitis of the zonular type. The limbus is vascularised and the vessels extend on to the corneal lesion. The iris is atrophied. Posterior synechiae and iris bombé are present.

remain in position, and it is possible that the lens might have been touched by the forceps.

A month later the lens began to shew opacities. Gradually it became opaque, the iris atrophied, and iris bombé occurred.

Nine months after operation the appearances were as in Fig. 10. The eye was shrinking; the cornea measured 12 mm. as opposed to 14 mm. for its fellow. There was a well marked superficial keratitis of the zonular type. The limbus was vascularised. Superficial fine vessels passed from the limbus to the patch of keratitis. The iris was much atrophied. There were posterior synechiae all around the pupillary ring, and some proliferation of iris pigment was noted on the anterior lens capsule. Iris bombé was present. There was a chalky white cataract.

Meanwhile events in the left eye had taken a somewhat different course. Here 6 pieces of 99.9 per cent. pure aluminium each weighing 0.83 mg. had been uneventfully inserted through a postero-superior Graefe puncture behind the equator. Gradually the corneal periphery became vascularised and the iris became atrophic. Lens opacities developed, more particularly in the posterior lens cortex. Some of the pieces of metal were covered first with white coating, and then gradually absorbed. Others shewed comparatively little change. Seven months after the initial operation the fundus appearances were as in Fig. 11. The disc

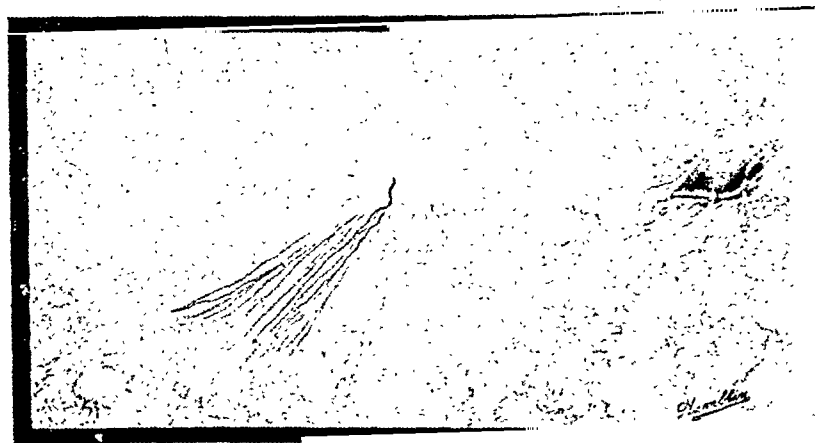


FIG. 11.

Rabbit No. 17, left eye. Implantation of 6 pieces of pure aluminium each weighing 0.83 mg. into the vitreous. Appearances as seen after 7 months. The disc is largely blurred by vitreous haze. Fibrous bands with arborescent blood vessels come forward into the vitreous. There is much pigmentary change. The retinal "imprint," marking the site formerly occupied by a bit of metal, is seen on a level with the disc.

was seen with some difficulty through a vitreous haze. There were fibrous bands with arborescent blood-vessels in the vitreous, constituting the picture of retinitis proliferans. The fundus was heavily pigmented, especially in its periphery. The degree of pigmentation was more than initially present, and was considered pathological. Near the disc there was a darkly pigmented patch at the site formerly occupied by a piece of metal. It was considered a retinal imprint.

The vitreous opacities gradually increased until at length the fundus was completely obscured. The iris slowly became depigmented above the pupil.



FIG. 12.

Rabbit No. 18, right eye. Implantation of 10 fragments of 0.83 mg. each pure aluminium into the vitreous. Appearances after 7 months. Four fragments of metal can be seen in the area shown in the illustration. A white film was present over some of them. One fragment is dotted with flecks of haemorrhage. A retinal "imprint" is seen just above the fragments; it is bordered by pigment. A fresh pigmented patch (not shown in the illustration) was present above the disc.

(c) Effect of 8.3 mg. in 10 fragments and of a single fragment of 6.64 mg. observed for 7 months in the two eyes of the same rabbit.

Rabbit 18. Ten pieces each weighing 0.83 mg. of 99.9 per cent. pure aluminium were inserted through a postero-superior Graefe puncture into the vitreous of the right eye. Gradually the corneal periphery became vascularised, and in the next few months the iris gradually atrophied, so that it transilluminated generally, with particularly atrophic patches near the pupillary margin. The lens developed streaky opacities. The fundus appearances 7 months after insertion of fragments are shown in part in Fig. 12. In the area shown in the figure 4 fragments of metal could be seen massed on the retinal surface. Some of the pieces were covered with a white film. One was dotted with flecks of haemorrhage. A retinal imprint could be seen as a greyish-white patch, bordered with a line of dark pigment. Elsewhere in the fundus (not shown in the figure) could be seen the scar of the insertion wound. There was a large fresh pigmented patch above the disc.

Meanwhile the effect on the left eye was tested by the insertion of a single piece of 99.9 per cent. pure aluminium in strip form weighing 6.64 mg. (equivalent to 8 of the 0.83 fragments used separately on the other eye). Gradually the cornea became vascularised peripherally and the iris atrophic. Lens opacities developed. Seven months later the fundus showed two large white retinal imprints which had been exposed by a recent shift of the metallic strip. A third imprint could be seen at the opposite end of the strip. In detail the appearances were those shown in Fig. 13 where the strip looks somewhat foreshortened. The strip was eroded and coated with white exudate. The two lower imprints were covered with white and necrotic material. The upper imprint showed fresh vascularisation at one side. A strand of gelatinous material ran to the junction of the two imprints. Powdery metal could be seen in the strand and scattered over the nearby fundus. There was pathological pigmentation in the fundus periphery.

(d) Effect of 20 mg. observed for 6 months.

Rabbit 23. 20 mg. of 99.9 per cent. pure aluminium were inserted into the right vitreous through a postero-superior scleral puncture. Six months later the cornea was vascularised and showed zonular degeneration. Other areas of cornea showed cholesterol crystal deposits in the stroma. The cornea was ectatic and only measured 9 mm. across as opposed to 13 mm. in the other eye. The appearance of the iris is shown in Fig. 14. The iris showed a central funnel-shaped depression with two vascularised white nodules projecting centrally in the pupillary area. The rest of the iris was in a condition of iris bombé and was much atrophied.

B.—INSERTION OF ALUMINIUM ALLOYS

(1) *Into the anterior chamber.*

(a) Effect of 0.5 mg. aluminium-copper (group 2), observed for 7 months.

Rabbit 12. A piece of aluminium-copper (group 2) weighing 0.5 mg. was inserted into the left anterior chamber. Eleven weeks

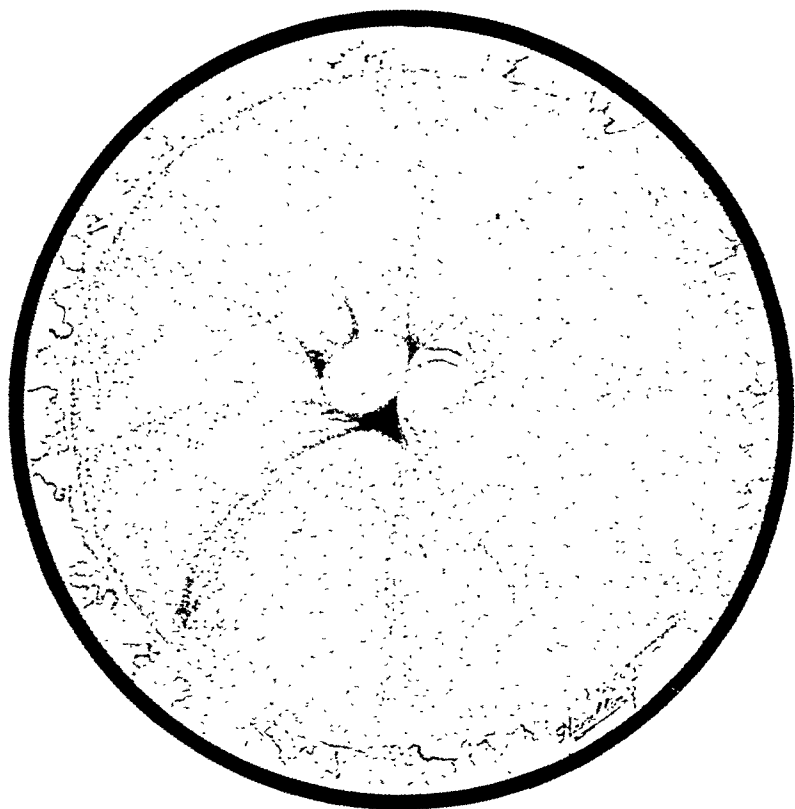


FIG. 14.

Rabbit No. 23. Implantation of 20 mg. of pure aluminium into the vitreous. Appearance of the iris after 6 months. The eye showed extensive degenerative changes. The cornea was ectatic, vascularised and developed zonular degeneration. Elsewhere in the cornea cholesterol crystals could be seen in the stroma. The iris, shown in the illustration, gave the appearance of a central funnel-shaped depression with two vascularised white nodules projecting centrally in the pupillary area. There was extensive atrophy and iris bombé was present.

later the appearance was as in Fig. 15. The limbus was somewhat vascularised. Above the pupil was a grey iris imprint. This was dotted with white-coated metallic powder from the fragment. Fine

capillary vessels could be seen in the superficial layers of the cornea opposite the imprint, though the latter did not seem to be directly vascularised. Near the pupillary margin lay other powdery fragments of the metal. There were posterior synechiae and some iris pigment on the anterior lens capsule. The lens showed streak, spiral and whorl opacities.

The main fragment lay below the pupil and adherent to the posterior corneal surface. The metal was partly absorbed and was encrusted with a yellowish-white covering. The cornea opposite the metal was slightly hazy and its superficial layers were vascularised.

Not long after this picture the metal again shifted, this time to near the pupillary margin. Here seven months after the insertion of the metal the appearances shown were those of Fig. 16.

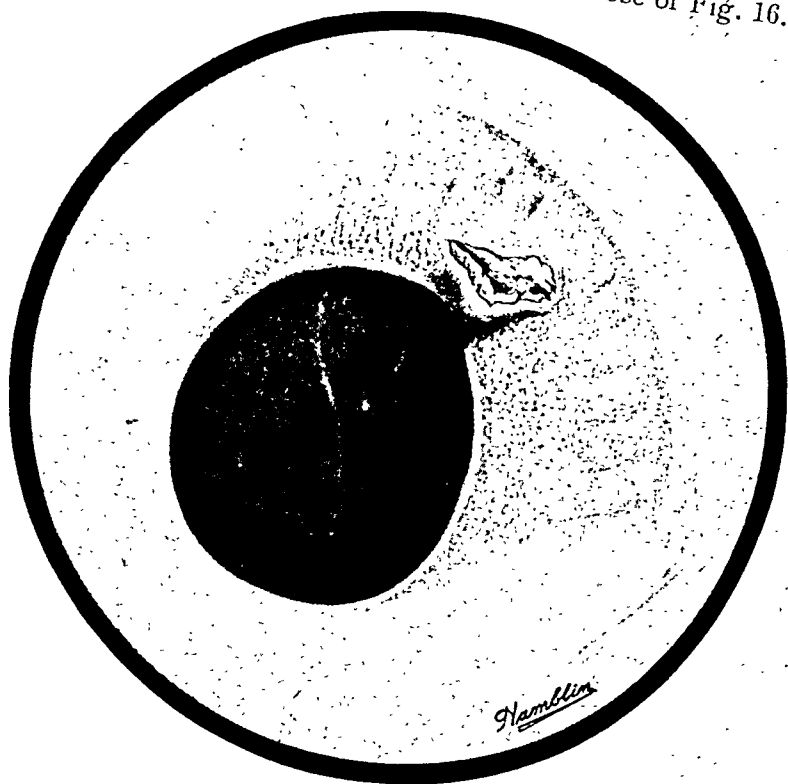


FIG. 17.

Rabbit No. 11. A fragment of aluminium-magnesium is adherent to the posterior surface of the cornea. The posterior surface of the metal is thickly covered by a white coating, which encroaches upon the corneal endothelium. The lens shows streaky cortical opacities.

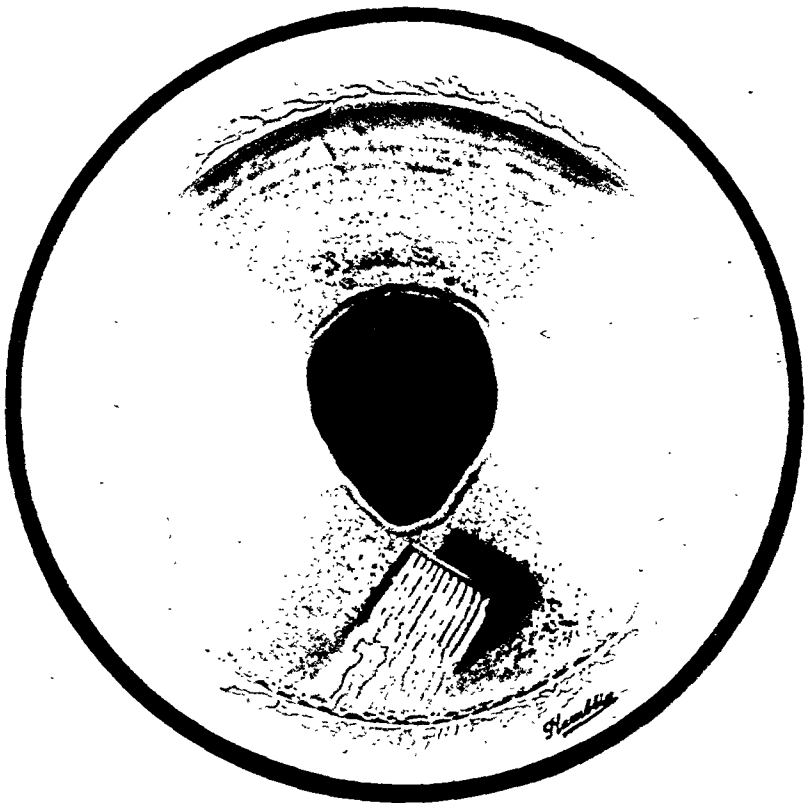


FIG. 15.

Rabbit No. 12. Insertion of 0.5 mg. aluminium-copper into the anterior chamber. Appearance after 11 weeks. The limbus is somewhat vascularised. A grey "imprint" dotted with white coated metallic powder from the fragment is seen on the iris above the pupil. The cornea above the "imprint" shows slight vascularisation. In the pupillary margin powdery fragments of the metal are seen. There are posterior synechiae and some iris pigment on the anterior lens capsule, and opacities are present in the lens itself. The metal itself is seen below the pupil. It is partially absorbed and encrusted with a yellowish white covering. The overlying cornea is slightly hazy and vascularised.

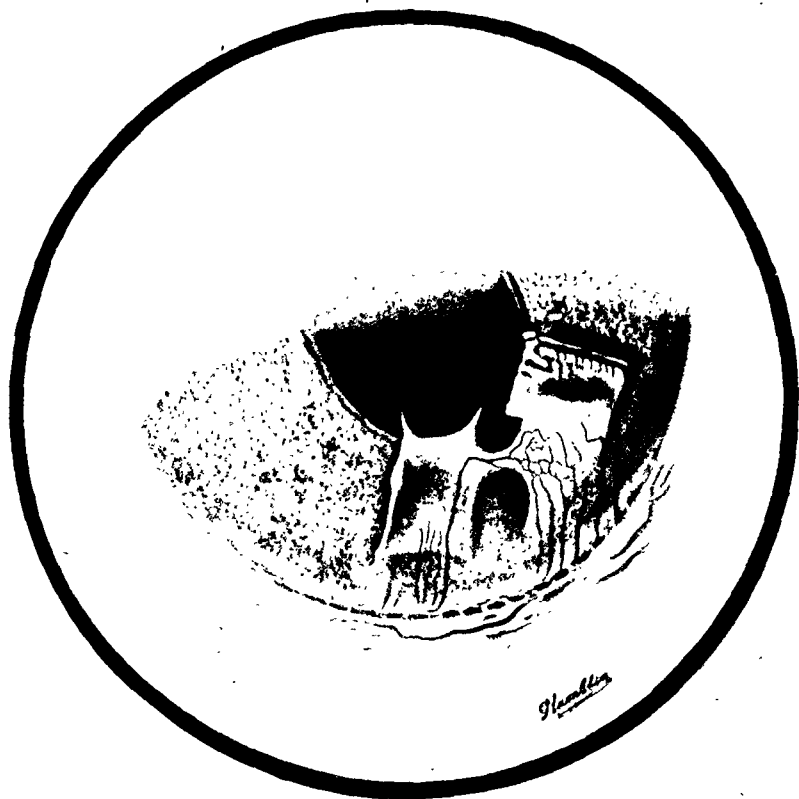


FIG. 16.

The same eye at 7 months. The fragment had shifted exposing an underlying "imprint" surrounded by fibrous tissue, which extends to the anterior lens capsule. The metal itself is in a more advanced stage of disintegration, and is covered by yellowish white coating. The lens shows opacities and polychromatic lustre. Some fragmentation of the metal can be seen.



FIG. 18.

Rabbit No. 1. An aluminium-copper fragment is seen in the anterior chamber. The fragment had fallen down upon the anterior lens capsule where there was a capsular opacity, also a superficial lens opacity. A broad pigmented synechia connected the opacity to the pupillary margin.

The metal itself was partly absorbed and fragmented. In part the metal showed a white coating. The limbus was vascularised. The corneal scar had healed.

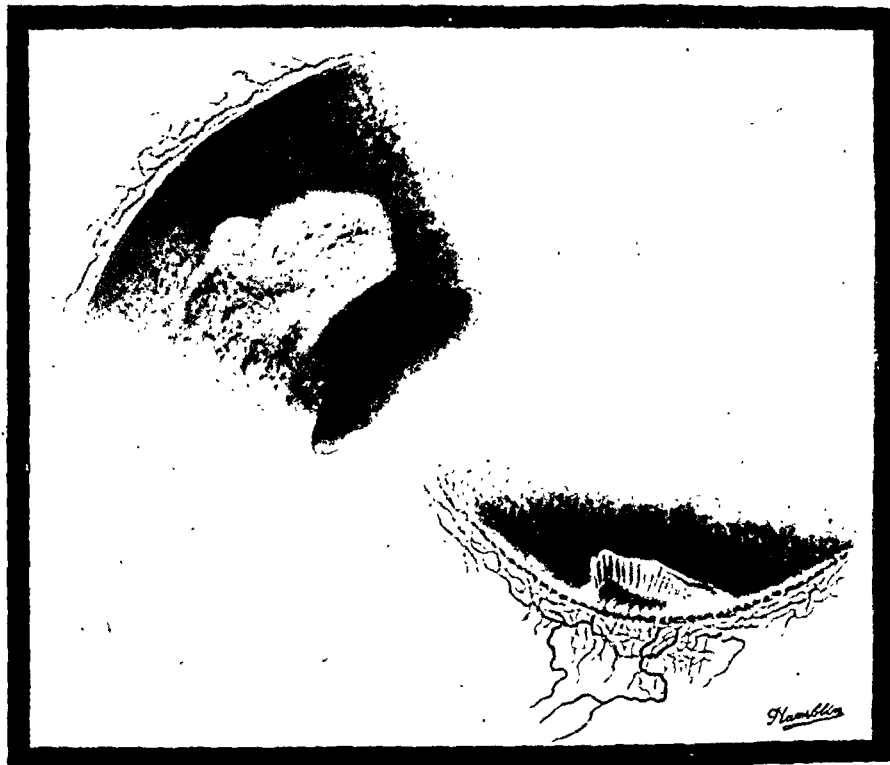


FIG. 20.

Rabbit No. 4. Insertion of 0.5 mg. aluminium-nickel into the anterior chamber. Appearances after 5 months. Above and anteriorly an iris "imprint" is seen. On its surface small fragments of powdery metal are present. The metal itself had sunk to the lower angle of the anterior chamber and is coated with yellowish white exudate. There is some limbal injection and fine capillary vascularisation of the superficial cornea of this area.

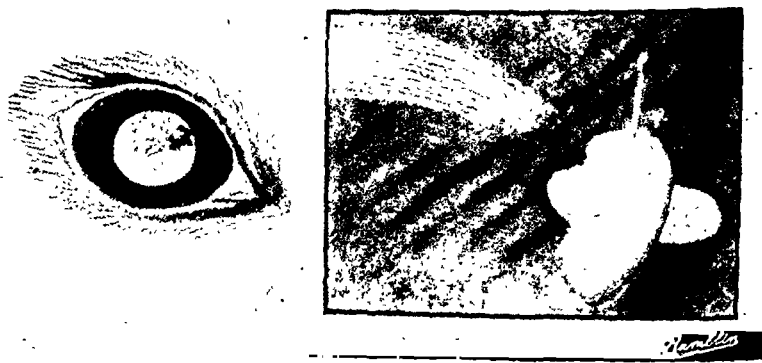


FIG. 21.

Rabbit No. 9. Insertion of 0.3 mg. aluminium-nickel into the vitreous. Appearances at three months. The lens shows posterior cortical opacities and some blood on its posterior surface. On the fundus the metallic fragment can be seen at right angles to an underlying "imprint," the site of its original position.

On the back of the cornea in the previous site of the metal was a faint grey imprint. Superficially in the cornea there was fine vascularisation in this region. The subjacent iris shewed a greyish-yellow imprint, surrounded on the iris by lines of fibrous tissue, which extended to the anterior lens capsule.

The cornea was superficially vascularised over the new site of the metal; and there was also a nebula. The metal itself lay at the pupillary margin and was dotted over with pigment. The fragment was mainly eroded and was covered with a yellowish-white coating. The lens shewed opacities and polychromatic lustre. The fundus shewed pigmentation thought to be pathological.

(b) Effect of 0.5 mg. aluminium magnesium observed for 9 weeks.

Rabbit 11. A fragment of aluminium magnesium (group 3) weighing 0.5 mg. was inserted into the left anterior chamber. The appearance 9 weeks later was as in Fig. 17. The metallic fragment was adherent to the posterior surface of the cornea. The area in contact with cornea was relatively bright. The posterior surface of the metal was thickly covered by a white coating. The lens shewed streaky cortical opacities. There was considerable fundus pigmentation.

Later the metal eroded forwards into the corneal stroma and gradually became fragmented. There were numerous lens changes, and gross fundus pigmentary changes.

(c) Effect of 0.4 mg. of aluminium-copper, observed for 18 weeks.

Rabbit 1. In this rabbit 0.4 mg. of aluminium-copper (group 6) alloy was inserted into the anterior chamber. Eighteen weeks later the appearance was as shewn in Fig. 18. There was limbal vascularisation. The corneal scar had firmly healed. The fragment had slipped down on to the anterior lens capsule, where there was a capsular opacity with some opacity of the subjacent lens matter. This opacity was connected by a broad pigmented and vascularised synechia to the pupillary margin. The metal was covered with a white coating, and was partially absorbed. Some fragmentation of metal had taken place, and white coated small fragments were visible on the surface of the lens opacity and on the synechia. Examination with the slit-lamp shewed polychromatic lens lustre, and there were pathological fundus changes.

(d) Effect of 0.4 mg. aluminium silicon observed for 3 months.

Rabbit 7. Here a fragment weighing 0.4 mg. of aluminium silicon (group 8) was inserted into the R. anterior chamber. Three months later the appearance was as in Fig. 19. The fragment was partially eroded and had a coat of powdery-white material with gelatinous material on the anterior surface peripherally. New

vessels rose from the iris in this region and passed across the front of the metal. The pupillary margin was slightly everted and adherent to the posterior surface of the metal. Dot and streak-shaped lens opacities were visible in the lens.

Later on the metal absorbed almost completely, an imprint shewed on the iris deep to the fragment, lens opacities increased, and the fundus shewed pathological changes.

(e) Effect of 0.5 mg. of aluminium-zinc with picture drawn at 5 months.

Rabbit 4. Five months after 0.5 mg. of aluminium zinc (group 9) had been inserted into the anterior chamber the appearance was as in Fig. 20. Above and anteriorly the iris shewed a greyish-

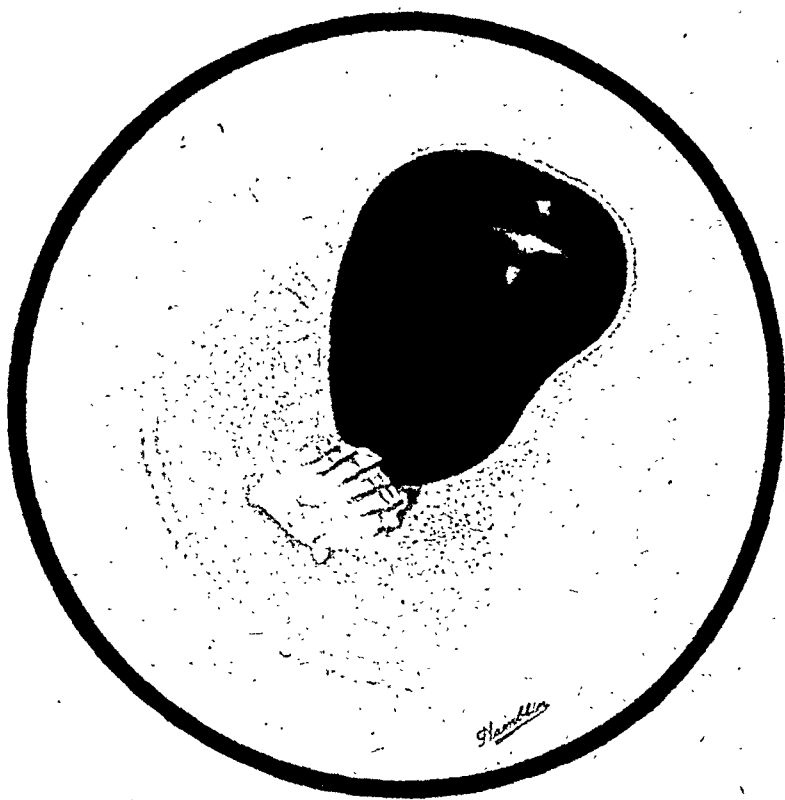


FIG. 19.

Rabbit No. 7. Implantation of 0.4 mg. aluminium-silicon into the anterior chamber. Appearances after three months. The fragment is partially eroded. Some fragmentation and formation of gelatinous material on its anterior surface is seen at the end away from the pupil. Iris vessels pass in front of the metal. The pupillary margin is slightly distorted, and adherent to the posterior surface of the metal. Opacities are present in the lens.

white imprint. On the surface of the imprint could be seen small fragments of powdery metal. There was slight vascularisation of the superficial cornea near the limbus. The actual metallic fragment had dropped to the lower angle of the anterior chamber, where it lay coated with yellowish-white exudate. There was enhanced vascularity of the limbus opposite the metal, and fine capillary vascularisation of the superficial cornea of this area.

The metal became deeply embedded in the iris, which was vascularised in this area. Peripheral pigmentary changes appeared in the fundus.

(2) *Into the vitreous.*

(a) Effect of 0.3 mg. aluminium-nickel-copper with picture drawn at 3 months.

Rabbit 9. A square fragment of aluminium—nickel-copper (group 4) weighing 0.3 mg. was inserted without difficulty into the vitreous through a scleral puncture (L. eye). There was no appreciable bleeding and the fragment was at once easily visible ophthalmoscopically. Three months later the appearances were as in Fig. 21. The lens shewed posterior cortical lens opacities. There was some blood on the posterior surface of the lens. Fundus examination shewed that the metallic fragment was completely coated with a whitish-yellow covering. The fragment had been dislodged by the animal's movements and lay at right angles to its original position. Deep to the fragment could be seen a retinal imprint at the original site.

Atrophic patches appeared in the iris. Lens opacities increased. The fundus shewed retinitis proliferans.

5.—Histological Findings

Twenty-nine eyes with intra-ocular foreign bodies and two control eyes were available for histological examination. The eyes were preserved in formol saline and later bisected for paraffin sections to be made. A serious difficulty was that the numerous small metallic bodies in the eyes rendered it difficult to cut sections. Even the minute fragments in many of the corneae were found to destroy instantly the cutting edge of the microtome knife, so that there was gross destruction and tearing of the surrounding tissues. This trouble was overcome to some extent by picking out any foreign bodies which were noted in the paraffin block; but this process in itself tended to spoil the ocular tissues immediately adjacent to the fragments, while it was usually found that some minute fragments were overlooked. In order to obviate this, many of the sections were cut rather thicker than usual. This helped to produce a complete section; but often at the expense of the finer histological details. Moreover, many of the sections were found with torn or folded corneae, damaged lenses, and other imperfections.

Much time was spent in endeavours to overcome these difficulties.

An attempt to work out a technique in which the metallic fragments might be removed by the use of some selective solvent, was unsuccessful. It seemed that the best course was to reject those parts of the sections adjacent to the foreign bodies as liable to be vitiated by accidental damage. Conclusions were based on sections somewhat distant from the fragments of metal.

In correlating the sections with the clinical observations on the experiments, it has to be remembered that a considerable time had elapsed in most cases between the original experiments and the acute immediate changes, and the final disposal of the animal.

The more significant histological changes

The cornea.—There was hyalinization in two cases (1, r and l). In three cases the section passed through the healed corneal scar (5, r; 20, l; 22, r). The cornea was frequently vascularised, completely sometimes (17, r; 23, r), and in other cases peripherally (17, l; 20, r). In two cases plaques were observed in the cornea (13, l; 23, r) their structure being vascular or fibrous. In one case (17, r) the cornea shewed peripheral wrinkling.

The anterior chamber shewed in two cases metallic fragments and exudate (14, r; 13, l) and in one case fibrous bands (13, l).

The iris.—A large number of pathological changes were observed. The iris shewed marked thickening in two cases (1, r; 7, l), in one case (1, L) there was round-celled infiltration with foreign body giant cells. In many instances the iris shewed atrophy, partial in 5 cases (4, r; 13, r; 16, r; 22, r; 23, l), well-marked in one (5, r), complete in 8 cases (11, r; 14, r; 14, l; 16, l; 17, r and l; 18, r and l). There was fibrosis in two cases (7, l; 17, l). Anterior synechiae were noted in 3 cases (14, l; 16, r; 23, r). Exudate was noted behind the iris in one case (17, r). Abnormal pigment distribution was seen in 10 cases; this was superficial in two cases (17, l; 22, r), patchy in three (4, r; 1, r; 16, r), and widespread and diffuse in five cases (5, r; 10, r; 13, r; 14, r; 17, r).

The lens.—As already stated the lens had frequently suffered in the cutting of the sections. In one case the lens seemed to be disorganised and calcified (9, l), and in 4 cases (9, l; 13, l; 17, l; 23, l) it was surrounded with exudate and incorporated with the ciliary body.

The vitreous chamber was filled with débris in one case (18, l).

The retina.—In three cases (1, r and l; 23, r) the retina was degenerate. There was loss of structure of the nervous elements in two cases (5, r; 11, l). Cystic degeneration was observed in the peripheral retina in two instances (7, r; 11, r). The retina might be atrophied in a local area (14, r), or more usually complete (8 cases: 5, r and l; 7, r; 8, r and l; 9, l; 10, r; 18, l). Fibrosis was observed in one case (9, l), organisation in another (7, l), and in one instance there was retinitis proliferans (17, l).

More active changes included choroidoretinitis (7, 1), cellular infiltration (10, r), active exudation in 6 cases (10, 1; 11, 1; 13, r; 14, r; 16, r; 23, 1), disorganisation in two cases (10, 1; 11, r).

The choroid.—Abnormal features were frequently seen. Exudate was noted in 4 cases (1, r and 1; 16, 1; 23, r). Thinning was noted in one case (5, r), and excessive vascularisation in another (13, r).

Optic nerve.—Degenerative changes were noted in one case (4, 1).

Taking the eye as a whole, although defects in the preparation of the sections prevented a complete review, the histological findings were explicit enough to shew that aluminium was not inert in the eye: it caused widespread low-grade inflammatory and degenerative changes, seen histologically as well as clinically.

Detailed histological reports are shown in Appendix II by Major J. O. Oliver.

Distribution of Aluminium in the Ocular Tissues

Since pathological changes from aluminium are widespread in the eye, and as it has also been noted that fragments of pure aluminium could apparently be completely dissolved in an eye (Case 22, r), the question arose as to how extensively the aluminium was disseminated through the ocular tissues.

It was at first hoped to utilise the intensely coloured red "lake" produced by aluminium salts with alizarinsulphonic acid as a means of tracing the aluminium, but consistent results could not be obtained.

A second method which utilises the red colour formed when aluminium salts combine with aurintricarboxylic acid was almost as disappointing. This colour reaction has to take place at a pH of 4.9 and is developed by heating at a temperature of 70—78 degrees C. This staining method proved uncertain at one time, producing red staining areas in the section, while subsequently similar sections from the same block, treated in apparently similar fashion, failed to stain. In the successful stainings the deeper layers of the cornea, the ciliary body, and the inner nuclear layers of the retina appeared to shew staining for aluminium most markedly. But the results were insufficiently constant to be considered a valid indication of the true distribution of the aluminium in the ocular tissues.

Whilst local changes near the fragments may be regarded as being directly due to the action of aluminium, it must be left an open question whether the more remote changes are due to aluminium possibly disseminated through the ocular tissues, or to some indirect toxic effect.

6.—Discussion.

Only scanty clinical records are available: three anterior chamber implantations (Jess, Fricke, Hesky), one anterior chamber and lens (Fricke), and two vitreous implantations (Savin). In one of Savin's cases the identification of aluminium was on strong presumptive evidence only. The animal case records are all upon rabbits and include 35 anterior chamber implantations (27 Savin, 8 Fontana) and 13 vitreous chamber implantations (9 Savin, 4 Fontana). The experimental work of Jess and Mielke is not recorded in sufficient detail for critical assessment.

There is close agreement between the animal and human case records. Jess's case shewed fragmentation of the metal, depigmentation of the pupillary margin near the foreign body, pigment deposition at the root of the iris, and a faint lens opacity. All these points were frequently demonstrable in the experimental animals. Fricke's first case shewed fragmentation of metal in the right eye, as frequently noted in the animals; his second case shewed fragmentation of the metal, lens opacities, and iridocyclitis; all these changes too were observed in the present experimental study. Hesky's case shewed corneal opacity, and iritis, both being changes found in the animal series. Likewise the changes in the vitreous observed by Savin clinically (coating of the metal, imprint formation, and some retinal pigmentation in one case, and more severe changes in the second: coating of the metal, mild iridocyclitis, vitreous opacities, localised imprints, and pigmentary changes in the adjacent retina) were also observed in the experimental animal. Whilst the changes observed in man so far—including changes in the metallic fragments, imprint formation and pigment deposition in adjacent ocular tissues—are paralleled in the experimental animal, the more remote changes seen in the rabbit have not as yet been noted in human eyes.

In the experimental series no tangible difference could be established between the effects of pure aluminium as opposed to aluminium alloys. Both groups give the same end result: lens opacities, quiet inflammatory iris changes, atrophy of the iris, choroiditis, choroidal and retinal degeneration and atrophy, retinitis proliferans, and also possibly vascularisation of various parts of the eye. There is some suggestion that with equal dosage, the presence of aluminium, or its alloys, is more deleterious when it is in the vitreous than in the anterior chamber. There is, however, no suggestion that particles in the anterior chamber are well tolerated. The size of the fragment seems to be a more definite consideration: the larger fragments are distinctly less tolerated than smaller fragments.

Aluminium and its alloys produce early effects in the adjacent tissues, but that widespread changes occur is clear from the almost

constant presence of fundus lesions, no matter what the size or position of the fragment be. Though aluminium and its alloys are not so destructive to the eye as iron and steel, these metallic intra-ocular foreign bodies call for operative removal. The technical difficulties for such removal are, however, such that it would seem that in individual cases an opportunist policy has to be adopted. It would seem that where the fragments are readily accessible, as in the anterior chamber, their removal should be attempted. Intravitreal fragments are probably best left alone except where their size is such that the loss of the eye is inevitable, if an expectant policy is adopted.

Summary

The literature of intra-ocular aluminium is reviewed. A case is reported in which a piece of "aluminium zinc" (or possibly zinc base aluminium) was observed over a period of three years as it lay on the retina of an only eye. The metal was at first bright and silvery. Later it became coated with a white cover, probably of aluminium hydroxide. The fragment shifted twice, each time leaving a retinal "imprint," before eventually disintegrating into white powder. A second case behaved similarly.

Twenty-five implantations of aluminium and aluminium alloy fragments were made into the anterior chambers of rabbits by a standardised technique; and 9 vitreous chamber implantations. There was no clinical difference in the behaviour of pure aluminium and various alloys. Doses varying from 0.3 mg. to 20 mg. were used; the ocular reaction tended to be more severe with the higher doses.

The fragments were observed to become coated with white powder, with yellowish exudate, with fibrin, or in late cases with jelly. A late change often seen was powdering and fragmentation of the metallic fragment. In 6 cases the fragment was completely absorbed.

Common local changes were necrotic "imprints" left by the metal; of these 6 imprints were corneal, 12 on the iris (9 grey, 2 white, 1 necrotic), 2 in the lens, 4 in the fundus.

General ocular effects on the eye included lens opacities in 28 eyes, striae, dots, vacuoles, and irregular opacities; these lens opacities were occasionally accompanied by polychromatic lustre. There were 5 anterior capsular opacities, 6 anterior cortical, 4 posterior cortical, 2 capsular imprints, and 3 complete cataracts.

Quiet inflammatory changes in the uveal tract were frequent. These included 10 cases of posterior synechiae, 2 cases of iris bombé, and 14 cases of iris atrophy.

Other changes included pathological fundus pigmentation in 23 cases, 6 cases of localised choroiditis and 3 of retinitis proliferans.

Pathological vascularisation, not reported in the literature in human cases, occurred often in the animals. It is uncertain whether the reaction is specific for aluminium.

Among other oddities were noted 2 cases of bullous keratitis, 1 staphylomatous eye (false buphthalmos), 2 cases of zonular keratitis, 1 interstitial keratitis, 1 deep keratitis, 2 pigmented corneae, 1 pigmented limbus, 2 cases of folds in Descemet's membrane.

Histological investigations were hampered by technical difficulties. The changes observed were such as would have been expected from the clinical findings. They included hyalinisation and vascularisation of the cornea, iritis and iris atrophy, abnormal iris pigmentation. Retinal and choroidal pathological changes were noted in many instances. Abortive attempts were made to trace the distribution of the aluminium through the tissues by staining reagents.

On the clinical and experimental evidence it is concluded that the eye is by no means inert to aluminium.

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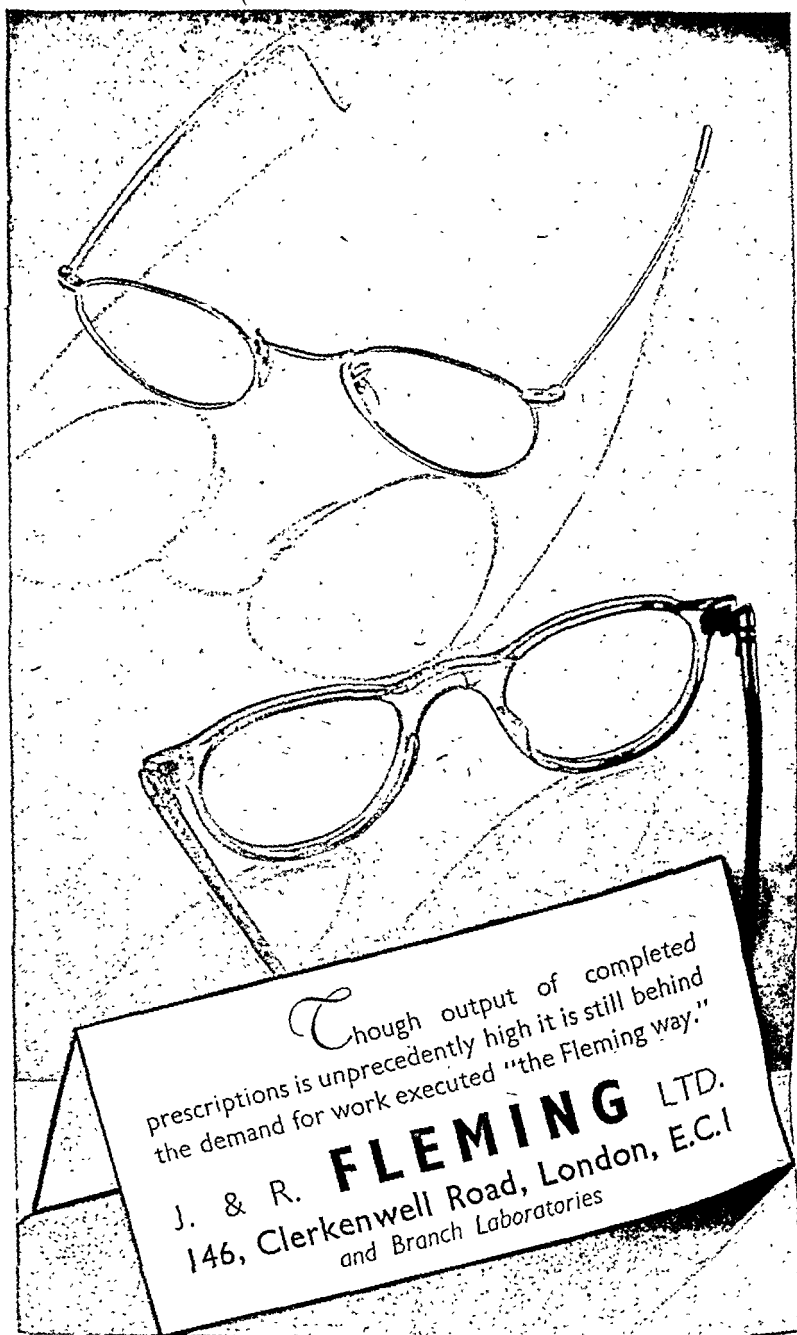


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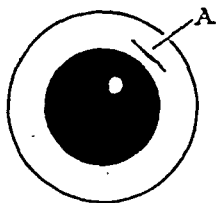
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APPENDIX I*

SUMMARY OF PROTOCOLS

RABBIT. N^o 1.

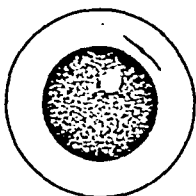
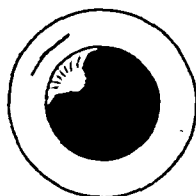
1. 27.9.42.



Left eye - An oblique incision was made through the cornea at A. - A piece of AlCu group 6 alloy (12% copper) was inserted by curved iris forceps. The same technique was repeated on the 'Right eye'

2. 28.9.42.

Right eye - shows faint exudate over the F.B. Entry wound is clear. A white film passes to the edge of the iris.



Left eye shows the F.B. lying on the lens capsule which is slightly opaque. Faint white film over F.B.

3. 4.1.43

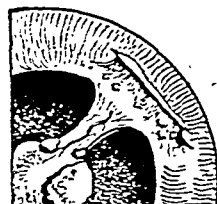
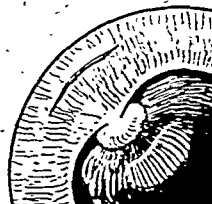
Right eye -
1. scar clear
2. metal coated
3. fibrous attachment to the iris
4. small opacity of the lens capsule.



Left eye -
1. anterior synechia to scar
2. white coat on metal
3. anterior capsular opacity
4. vascularisation of fibrous adhesion of metal to iris

4. 7.1.43.

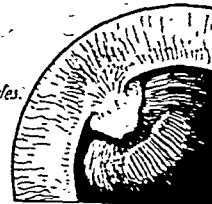
Right eye -
1. scar clear
2. metal coated & embedded in anterior capsule.
3. peripheral chorio-retinitis



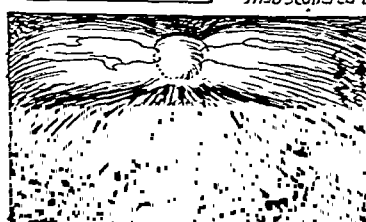
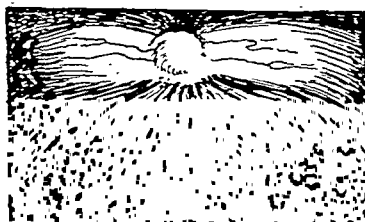
Left eye -
1. anterior synechia to scar with small glistening F.Bs in it
2. ant. capsular lens opacity with the F.B. on it and several scattered pieces of metal
Iris discoloured. Scattered jelly over F.B. - folds in Descemet's

5. 16.5.43.

Right eye -
1. there is bullous keratitis and fine keratic precipitates.
2. white mass at site of F.B. iris adjacent to it
3. imprint in the lens
4. there are some necrotic areas in the fundus at about 4 o'clock.



Left eye -
1. a few corneal bullae
2. there are folds in Descemet's
3. the metal is crumbling, surrounded by lens opacity
4. the fundus shows more pigmentation, gross mass posteriorly to disc. Also scattered white patches



* The protocols on Rabbits Nos. 6, 9, 15, 19, 20, 21 and 24 are devoid of illustrations and appear textually after the protocol on Rabbit No. 23.

RABBIT N^o 2.

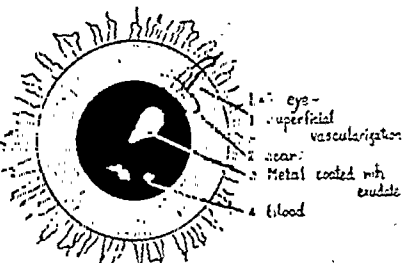
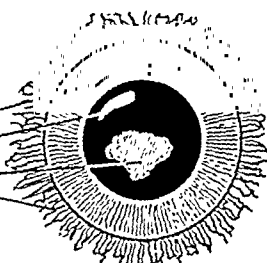
5.10.42.

*Under cocaine anaesthesia 1.5 mg. of 98% aluminium was inserted into R. eye by previous technique
Similarly 0.3 mg. of 98% aluminium was introduced into L. anterior chamber.*

① 10.10.42.

Right eye -

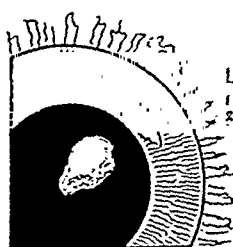
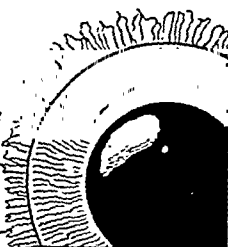
1. Iris in scar
2. F.B coated with yellow exudate
3. Hyphaemia
4. Ciliary injection



② 4.1.43.

Right eye -

1. Scar - clear
2. metal coated with white exudate and adherent to back of cornea
3. iris pigment on lens
4. There are large whitish vitreous opacities and some retinal pigment changes



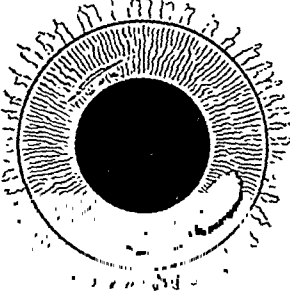
Left eye -

1. Scar - now white
2. Metal - white coated. Opacities in lens immediately beneath 2

③ 6.1.43

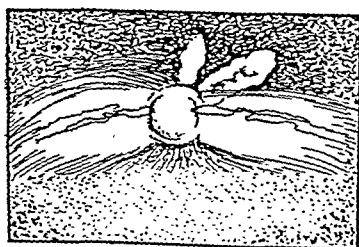
Right eye -

1. Scar pigmented
2. metal on iris
3. free fragment in A.O.
4. vascularized corneal imprint containing sr metallic fra.

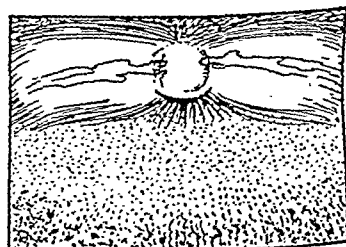


Left eye -

1. Corneal scar shows thread-like anterior synechia
2. Fragment is discoloured and sunk into anterior lens capsule, surrounded by fibrinous exudate.



Fundus - patches of choroiditis above macula
Some peripheral pigmentation



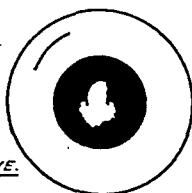
Mottled pigmentation of peripheral fundus

RABBIT N^o 3.

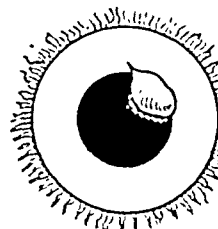
Cocaine anaesthesia was employed. The R. eye was dislocated outwards and 0.3 mg. group 2. AlCu alloy was inserted without difficulty. The rabbit started when the left incision was made, there was an iris prolapse. The 0.3 mg. group 2. F.B. was fairly easily inserted. The prolapsed iris was snipped off. There was a small vitreous loss.

13.10.42.

Foreign body on anterior lens capsule surrounded by exudate.



RIGHT EYE.



Large iris prolapse covered with fibrin.

The eye appeared injected.

LEFT EYE.

RABBIT N^o 4.

17.10.42. Anaesthesia by injection of 0.75 cc. of 1gr per cc. nembulal intravenously into the vein of the L. ear.

Cocaine anaesthesia was employed as well.

R. eye - a 0.4 mg. fragment of group 9 AlZn was inserted through a straight keratome incision.

L. eye - a 0.5 mg. of group 9 AlZn was inserted through a Taylor knife incision with no difficulty.

9.1.43.

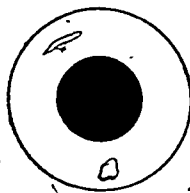
Thread-like anterior synechia.

A few metallic flakes embedded in the corneal stroma.

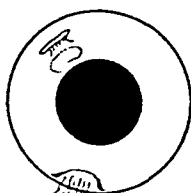
The metal fragment has dropped across the anterior chamber.

It is seen below, dashed with a few patches of iris pigment.

Fundus shows pigmented patches at 7 and 4 o'clock.



RIGHT EYE.



Corneal scar shows a few flakes of metal.

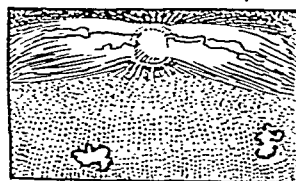
Fine anterior synechia imprint on the iris.

The metallic fragment lies partly coated in the angle of the anterior chamber, at 7 o'clock.

The cornea is vascularized superficial to the fragment.

Fundus - peripheral changes below.

(LEFT EYE).

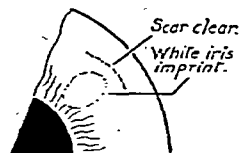
RABBIT N^o 5.

25.10.42. Anaesthetized intravenously by injection of nembulal solution gr.1.1cc. 0.5 mg. fragments of pure 99.9% aluminium were inserted by the usual technique into the A.C. of each eye.

9.1.43.

Left eye (DIAGRAM). Scar clear. White imprint on the iris at the site of the absorbed metal - Vitreous opacities. - Peripheral fundus changes.

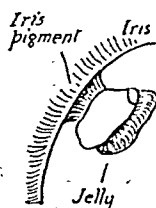
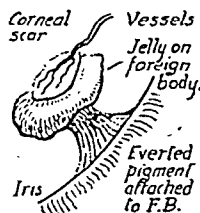
Right eye - Corneal scar is pigmented, with fine synechia. The metal lies faintly coated, in the angle of the anterior chamber below. There are peripheral choroidoretinal changes.



RABBIT N° 7.

B.11.42. Anaesthetised by intravenous injection of Icc. of gr1 ad Icc. nembulal. By the usual technique two fragments each of 0.4 mg. aluminium silicon groups were inserted into the anterior chamber of each eye.

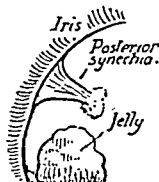
1. Right eye - 9.1.43
Corneal scar is vascularised. FB lies on the ant. lens capsule surrounded by gelatinous material. A line of pigment runs from the everted iris margin to foreign body. There are cortical lens opacities and peripheral fundus changes.



Left eye - Corneal scar is clear. Metal fragment lies on the anterior lens capsule, presenting an iridescent appearance. There is jelly on the front of the metal. Iris margin is everted with pigment on the lens capsule and foreign body. Some opacity of lens. Peripheral mottling of fundus.

2. Left eye - 16.5.43.

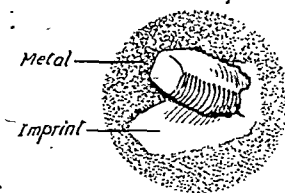
The scar is clear. There are posterior synechiae. The metal seems completely absorbed. There is jelly in the anterior chamber. The lens shows opacities.

RABBIT N° 8.

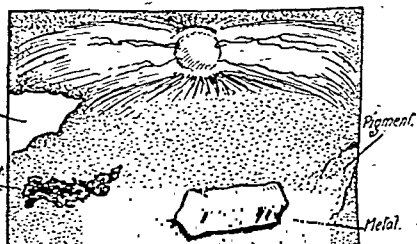
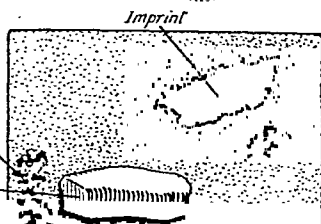
B.11.42. Anaesthetised by intravenous injection of Icc. gr1 ad Icc. nembulal.

Right eye - A Groese knife scleral puncture was made in the 2 o'clock meridian behind the equator of the globe. A piece of pure aluminium weighing 0.9 mg. was inserted with forceps.

Left eye - A scleral puncture was made in the 10 o'clock meridian behind the equator. 0.9 mg. aluminium was inserted.



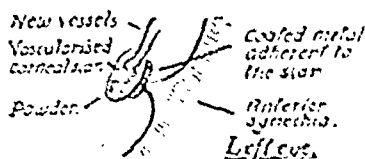
9.1.43. Right eye - Slit-lamp shows a few lens striae. Fundus examination shows the metallic fragment far forward with an iridescent white coat. Posterior to the fragment the retinal background shows a pigmented scar thought to be a retinal imprint. Fundus shows pigmented mottling.



L. eye - The entry scar is plainly seen with its pigmented edges. The metal, covered with a white coat, lies some way below the disc - it has an iridescent appearance. A pigmented scar lies posterior to the metal, having the appearance of an imprint. There are other pigmented patches near the metal. - Peripheral mottling of the fundus.

RABBIT N° 11.

27.11.42. The rabbit was anaesthetised by intravenous injection of nembutal, using the usual technique. A fragment of AlMg group 3 weighing 0.5 mg. was inserted into the anterior chamber of each eye by the previously used method.

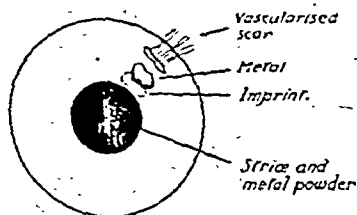


17.1.43. Right eye - Metal fragments in the wound scar. Also an anterior synechia. The main mass of the metal is adherent to the scar posteriorly. It has a yellowish white coating. There are cortical lens striae and vacuoles. - Marked peripheral pigmentation in fundus.

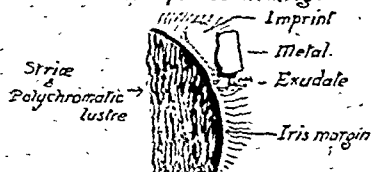
Left eye - The corneal scar is vascularised; there is metallic powder in the scar. The main mass of the metal is adherent to the scar (see diagram above). The metal is covered with a white and yellow coat. - There are cortical lens spouters and considerable fundus pigmentation.

RABBIT N° 12.

27.11.42. The animal was anaesthetised by intravenous nembutal into the marginal vein of the ear. Pieces of 0.5 mg. weight AlCu group 2 were inserted into both anterior chambers by the usual technique.

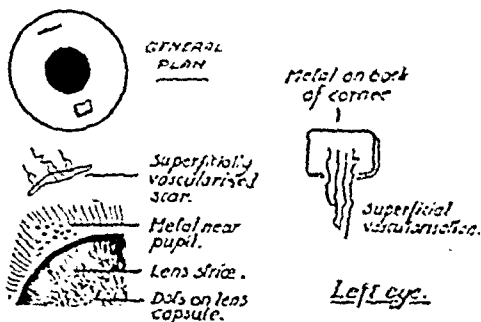
Right eye. 23.1.43

The corneal scar is vascularised. The metal is covered with exudate posteriorly. There is a necrotic area in the iris underneath the metal. There is an adhesion between the iris and the metal. Powdered metal lies on the lens capsule in the pupillary area. - The lens shows striae. There is considerable fundus mottling.

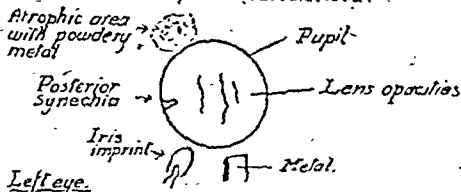
Right eye. 30.5.43.

The metal, covered with exudate, lies near the pupillary margin. It has shifted slightly away from the imprint. Dots, streaks and striae on the lens. Polychromatic lustre on slit lamp examination.

Fundus changes are more marked and thought to be pathological.



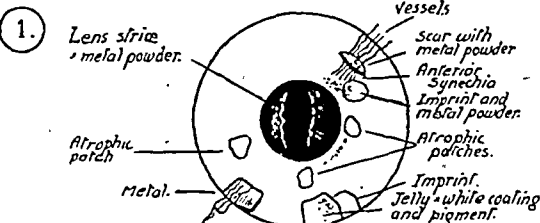
Left eye. Superficial vascularisation of corneal scar; powdery fragments in scar. - Posterior synechia and lens opacities. Some small metallic fragments lie on the anterior surface of the iris. The main metal is adherent to the posterior surface of the cornea. The anterior surface of the metal seems infolded; but the cornea over the surface is vascularised.

Left eye.

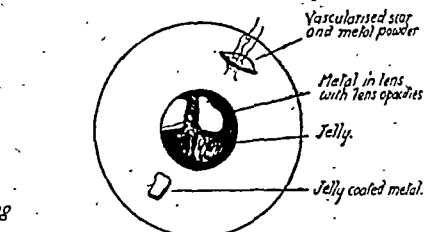
Atrophic area of iris on anterior surface above. This area shows powdery metal. Vascularised iris imprint below the pupil, from which the metal has shifted again laterally. There are posterior synechia and lens striae. Some patches, thought to be pathological, are shown in the fundus.

RABBIT N° 13.

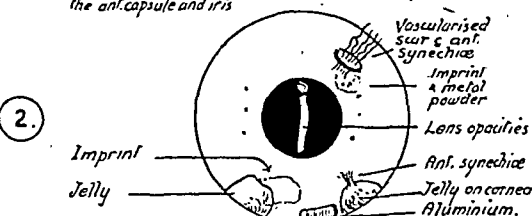
21.6.43. The animal was anaesthetized by 0.75 cc. Nembutal solution. Through posterosuperior corneal incision 4 mg. of pure Al (99.9%) was inserted into the anterior chamber in 1 mg. fragments. A similar quantity was inserted into the right eye, but using an anterosuperior incision.



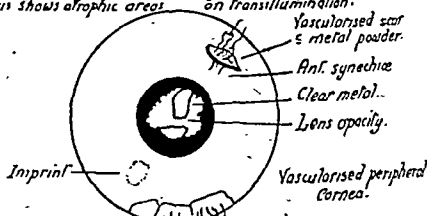
27.6.43 - Right eye. - Corneal scar is vascularised and contains some powdered metal. Ant. synchia near the base of which is an iris imprint. Lens striae and al powder on the ant. capsule and iris.



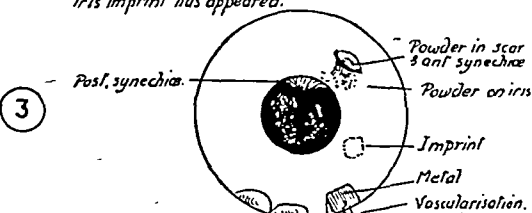
Left eye - Vascularised powdered scar. Two pieces of metal have sunk into the lens, and are surrounded with opacities. Covered with jelly. At lower angle of A.C. lies jelly coated metal. Iris shows atrophic areas on transillumination.



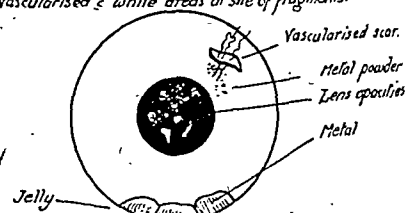
26.9.43. Lens opacities more pronounced. An ant. synchia passes from one of the fragments through the cornea. Jelly on the post. surface & in lower angle of the A.C. - A fresh iris imprint has appeared.



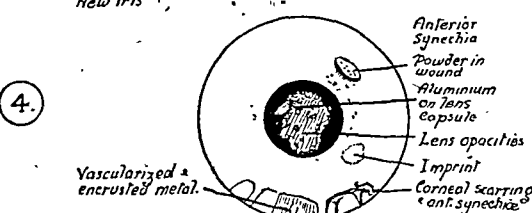
Two fragments of 4 metal have sunk into the lens. Their surface, as far as can be seen is shiny and clean. Imprint on Ant. iris surface at 7 o'clock. Lower cornea is vascularised & white areas at site of fragments.



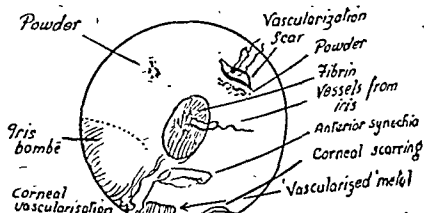
30.10.43. Iris more advanced. New iris.



Much of the metal in the lens has been absorbed. There is more opacity of the lens.



5.2.44. More pigmentation at ant. end of op. nerve fibres. A disc. Transillumination shows much iris atrophy. Above diagram shows still more corneal scarring & anterior synchia.



Scar more vascularised. Pupil closed by fibrin. New vessels on ant. of iris extend into pupillary area. Large ant. synchia below pupil. Much corneal scarring. At 3 o'clock there is an iris bombe.

7.5.44. Right eye - No metal left in lower AC. Some jelly remains - rather more fundus changes.

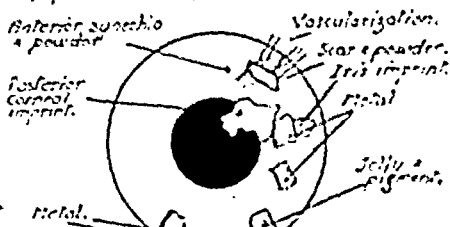
Left eye unchanged but for more vascularisation of cornea.

RABBIT N^o 14.

21.6.43. Intravenously applied 0.5cc nembutal solution anaesthetized. Through the usual corneal incisions 6 mg. of pure (99.9%) aluminium was inserted in 1mg. fragments into each anterior chamber, without particular difficulty.

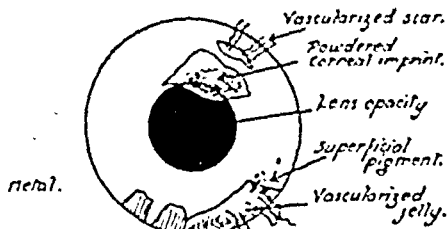
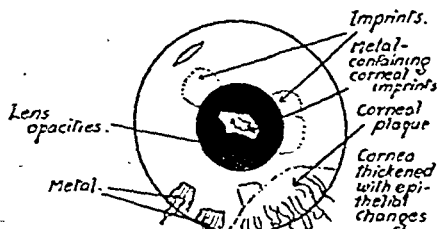
Right fundus — Some speckled pigment. Dark patches in the 6 & 7.5 meridians.

Left fundus — Dark patches in 6 & 4.3 meridians.



8.8.43 — RIGHT EYE. — Scar contains powdered metal. Multiple iris imprints, and a central corneal imprint which encloses a fragment of the metal. Several of the bits lie in the lower angle of the anterior chamber. They are covered with fine vessels; some are white coated with spots of pigment. — There are many lens opacities. — Fundus shows pigmented patches.

Left eye — The corneal scar is heavily vascularized and incorporates much metal powder. Anteriorly it also contains metal powder. There is a posterior corneal imprint, also an iris imprint. Some of the pieces are bright and others entrapped and coated with jelly and pigment.



5.2.44 — RIGHT EYE — The main fresh feature is a vascularized corneal plaque below. The cornea is thickened with epithelial changes.

Left eye — The main features are illustrated in the above diagram.

RABBIT N^o 16.

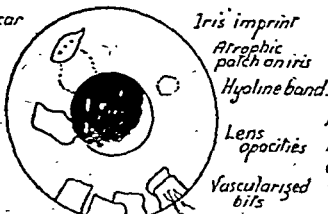
27.6.43. Anaesthetized by 0.75cc. nembutal intravenously. 10 pieces each of 1 mg. 99.9% pure Aluminium were inserted by the usual technique into the right A.C. 8 into the Left.

Right fundus showed patches of pigment near the disc in the 6, and 8 o'clock meridians.

Left fundus showed infero-lateral pigmentation.

Vascularised scar with powder.

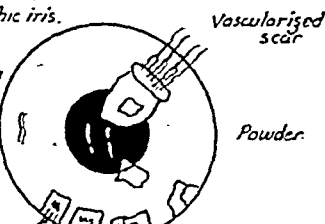
Posterior corneal imprint.



Atrophic iris.

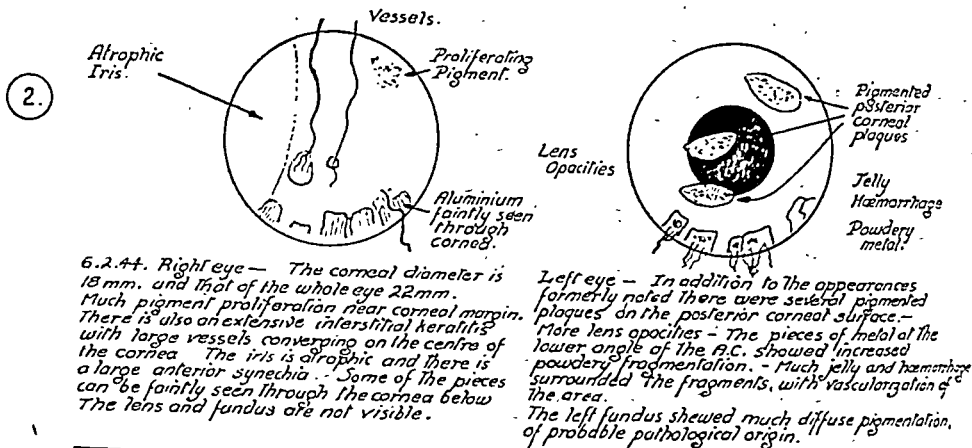
Anterior Synechia

Lens Striae & poly-chromatic lustre

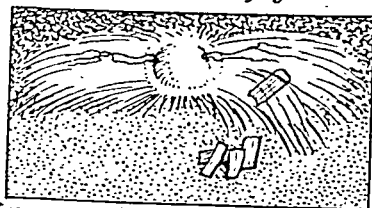


Right eye — vascularised corneal scar containing powdery metal. Iris imprint near entry scar. Large posterior corneal imprint. Ant. border of iris shows atrophic patch. Several metallic fragments in lower angle of A.C. while coated & vascularised. Bits of striae in lens. Hyaline band across pupil. Ready transillumination — Increased fundus pigment.

Left eye — A large mass of jelly covering a piece of metal lay behind the much vascularised corneal scar. — A second piece of metal was adherent to post. corneal surface. Metallic powder on back of cornea. More jelly post. to metal. Another piece of metal post. cornea, below pupil. — Several pieces coated with jelly in lower angle of A.C. — Several hemorrhage patches on metal. — Iris much atrophied. — Lens lustre & striae.

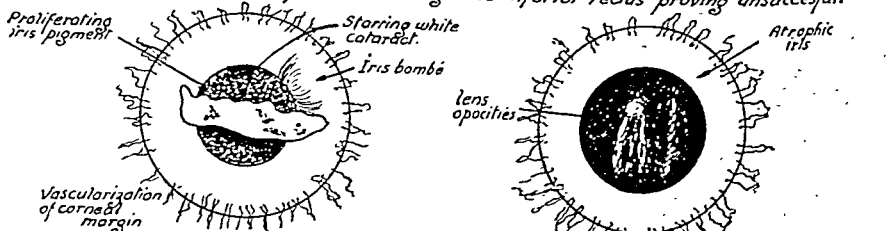
RABBIT N° 16. (Continued).RABBIT N° 17.

4.7.43. Anaesthetised intravenously by 0.75 nembutal solution.



Right eye — A scleral puncture was made with the Graefe knife in the anterosuperior quadrant of the eye posterior to the equator. 4 fragments of 99.9% pure Aluminium each weighing .23 mg. were inserted into the vitreous via a puncture which passed through the anterior bundle of optic nerve fibres — There was much difficulty in making the pieces stop in position.

Left eye. — 6 identical pieces were inserted into the vitreous through a postero-superior Graefe puncture, a previous puncture through the inferior rectus proving unsuccessful.



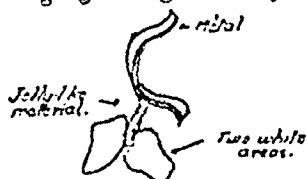
RABBIT N^o 18.

4.7.43. Anaesthetised by 0.5cc nembutal solution intravenously.

Right eye - A postero-superior Graefe knife puncture was made.
10 pieces, each weighing 0.85mg of pure Aluminium were inserted with curved forceps into the vitreous. There was some difficulty as the vitreous was apt to herniate.

Left eye - A postero-superior Graefe puncture was made.

An unsul strip of 8 pieces of 99.9% pure Aluminium each weighing 0.85mg. was easily inserted into the vitreous.



7.11.43 - Some vascularization of the deep peripheral stroma of the cornea. Atrophic iris patches. Some small lens opacities.

Ophthalmoscopically the pieces were massed together postero-superiorly in the vitreous.

The surface of the metal was iridescent, some pieces were coated with a white film.

Postero-superiorly on the retina was a white film. This was partially vascularized but separate from the entry scar. Fundus otherwise normal except for one or two white patches.

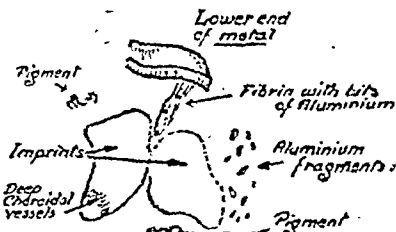
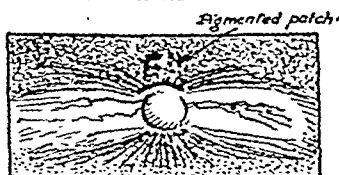
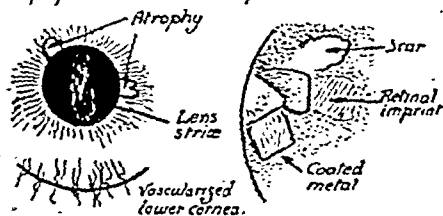
Peripheral corneal vascularization. Iris atrophy. Lens opacities were present.

Fundus - A long curved metal fragment could be seen with a faint white coating.

Postero-inferiorly were two white areas apparently covered with necrotic material.

Near the lower end of the metal was a mass of jelly-like material extending between the metal and the retina. - The fundus elsewhere was extensively mottled with white patches.

Postero-superiorly the entry scar could be seen with pigmentation around. The upper half of the metal was also surrounded with jelly.



10.2.44. (Above diagrams).

Vascularized corneal periphery, particularly below. - Iris transilluminated generally, particularly at some atrophic patches below. Streak-like lens opacities. Retinal imprint unchanged - metallic fragments perhaps more coated. - Pigmented patch above disc.

Left corneal periphery was vascularized.

Iris transilluminated freely. - Several especially atrophic patches.

Retinal imprint near the upper end of metallic fragment. The lower end of fragment had shifted. The metal was coated and a strand of fibrin ran to the two retinal imprints previously noted. - Some powdery metal was lying in the strand.

One imprint showed some deep vessels running in the choroid and pigmentation alongside.

There were some scattered aluminium fragments near the imprints and some peripheral pigmentation in the fundus below.

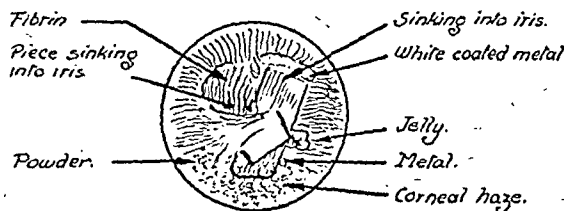
7.5.44. Slit lamp appearances unchanged. More pigmentation around retinal imprints.

7.5.44. Pigment above disc (see above) was more marked - metal imprints, no change.

RABBIT N^o 22.

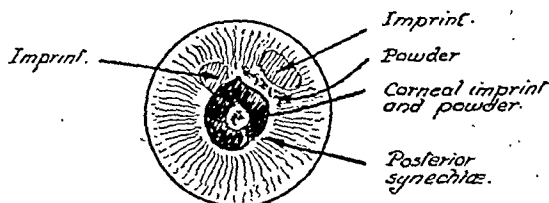
10.10.43. Anaesthetised intravenously by 0.5cc nembutal solution. Through a postero superior corneal puncture 12 mg. of 99.9% pure Aluminium was inserted in two 6mg. strips into the Right A.C.

1.



7.11.43. Right eye — The two strips, as shown above, were sinking into the iris. An oblique strip was covered with a fibrous material save for the lower end which impinged on the cornea. The cornea was hazy in this area and there was a deposition of gelatinous material near the end of the piece of metal. The vertical fragment showed sinking into the iris at the upper end. In this region the metal was covered with white deposit.

2.



6.2.44. The two pieces had completely disappeared save for some metal powder on the anterior iris surface. Two obvious imprints remained on the iris surface. There was a corneal imprint sprinkled with powdery metal.

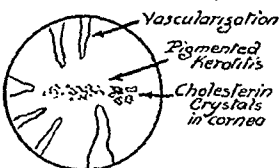
RABBIT N^o 23.

10.10.43. Anaesthetised intravenously by 0.5cc nembutal. 20 mg. of 99.9% pure Aluminium were inserted into the Right vitreous by a posterior scleral puncture.

1.

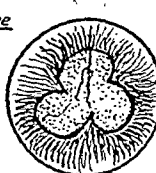
Right eye
10.2.44.

Corneal vascularization with zonular keratitis



Right eye

Lens opaque with vascularization of the anterior lens capsule



Vascularization.

Lens opacities.

Iris bombe.

2.

Right eye
11.5.44.

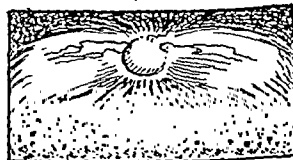
Cornea ectatic, heavily vascularized

Pigment on the anterior surface A.C. deep + iris funnel shaped.

White nodule projecting centrally in pupillary area



Eye somewhat shrunken. Corneal diameter of 9mm (L. cornea = 13mm)



Fundus mottling was noticed in each eye before the experiment but appeared to have increased in the left eye on examination 11.5.44.

RABBIT No. 6.

8.11.42. An injection of 2 c.c. of gr. 1 ad 1 c.c. nembutal solution was given intravenously into the marginal vein of the ear. The rabbit was rather quiet; but the heart was beating at the start of the experiment. 0.4 mg. aluminium-silicon (group 8) was inserted in the right anterior chamber by the usual technique. The rabbit was found to be dead. It was thought that the death was due to an overdose of anaesthetic. No difficulty appeared in technique etc. during the experiment otherwise.

RABBIT No. 9.

22.11.42. The animal was anaesthetised with 1.75 c.c. nembutal solution intravenously. No difficulty was experienced.

RIGHT EYE. A scleral puncture was made with the Graefe knife accidentally in the 3 o'clock meridian when the animal sneezed suddenly. Afterwards a deliberate puncture was made in the 1.0 o'clock meridian behind the equator. A small bead of vitreous protruded. An attempt was made to insert 0.3 mg. of Group 4 (AlNiCu) alloy. The fragment stuck subconjunctivally. However it was eventually retrieved and thrust with the forceps intrasclerally. It was assumed it was now in the vitreous; but this was difficult to verify ophthalmoscopically as there was some haemorrhage into the eye at the site of the puncture.

LEFT EYE. A scleral puncture was made with the Graefe knife at 10 o'clock meridian behind the equator. A square 0.3 mg. fragment of Group 4 (AlNiCu) was inserted without difficulty and verified in the vitreous ophthalmoscopically.

17.1.43. **RIGHT EYE.** There were lens opacities. Haemorrhages were seen on the back of the lens and in the vitreous. The metallic fragment had a white coating. The rest of the fundus was normal as far as seen.

LEFT EYE. There were subcortical lens opacities. These shewed polychromatic lustre as seen with the slit-lamp. The metal could be seen covered with a white coating. There were vitreous opacities and bands in the vitreous suggesting retinitis proliferans.

30.5.43. The **RIGHT EYE** shewed some atrophic iris patches. In addition to the lens opacities the right eye shewed polychromatic lustre in the lens. A collection of blood could be seen on the back of the lens. There was retinitis proliferans above in the fundus.

The **LEFT EYE** shewed iridocyclitis with massive keratic precipitates. The iris stroma had atrophied so that vessels shewed through. The pupil was occluded by vascularised fibrinous exudate.

27.6.43. The animal became mated.

16.9.43. The animal was killed by the air-embolism method and its eyes enucleated.

RABBIT No. 10.

22.11.42. The animal was easily anaesthetised by 1 c.c. of the nembutal solution intravenously in the marginal vein of the ear. A fragment of Group 4 (AlNiCu) was inserted into each eye without difficulty, the fragment being put into the anterior chamber.

24.1.43. The rabbit had disappeared.

30.5.43. The rabbit was discovered mixed up with other laboratory stock.

RIGHT EYE. There was a pigmented corneal scar with an anterior synechia attached. The lens shewed opacities. The metallic fragment could not be seen. There was gross fundus pigmentation and vitreous opacity.

LEFT EYE. The corneal scar was pigmented. There were anterior synechiae. There were some lens opacities, many vitreous opacities, and much pigmentation. The metal, partially dissolved and very thin, lay below in the anterior chamber.

10.10.43. The rabbit was killed and its eyes enucleated.

RABBIT No. 15.

This was a silver-grey doe weighing 4lbs. The right ear had been bitten badly; the left shewed a smaller bite. The right fundus was rather light with pig-

mented patches at 6 and 8 o'clock meridians. The left fundus showed similar patches at corresponding meridians 4 and 7.

27.6.43. The animal was anaesthetised with 0.5 c.c. nembutal solution intravenously. Two pieces, each 1 mg. in weight, of pure (99.9%) aluminium were inserted into each anterior-chamber by the usual technique. There was some haemorrhage into the right anterior chamber. The rabbit recovered and hopped around its cage. Later that day for no apparent cause it was found dead in its cage.

RABBIT No. 19.

This was a black-and-white buck of approximately old English type.

19.9.43. RIGHT EYE. Five fragments of about 1 mm. square of aluminium foil were inserted into the upper fornix of the conjunctiva. Six hours later the material had worked out of the conjunctival sac leaving the eye normal.

LEFT EYE. Approximately 1 mg. of scrapings from the ribbons were inserted. This seemed to be a mixture of paper, graphite, and aluminium. Six hours later all the material had worked out of the conjunctival sac, leaving the eye normal.

26.9.43. The animal was satisfactorily anaesthetised by 0.75 c.c. of nembutal solution intravenously. A posterosuperior corneal puncture was made in the right eye, and 3 cm. of coiled up platinum wire, weight 15 mg. were inserted into the anterior chamber without difficulty.

The animal was watched for about three weeks during which period the metal seemed to be completely inert. It was then accidentally killed with some other laboratory stock, and the eye was lost.

RABBIT No. 20.

This was a brown old English buck. Fundi pigmented below disc.

19.9.43. RIGHT EYE. Five pieces of R.A.F. aluminium foil each 1 mm. square were inserted into the upper conjunctival fornix. Six hours later all the material had worked out of the conjunctival sac, leaving the eye normal.

LEFT EYE. 1 mg. of foil scrapings (graphite, paper, and aluminium) were inserted into the upper conjunctival fornix. Six hours later the material had all worked out of the conjunctival sac, leaving the eye normal.

26.9.43. The animal was anaesthetised by 0.5 c.c. of nembutal solution intravenously. The animal was quiet under the anaesthetic; but gave two high pitched reflex squeals.

A posterosuperior corneal puncture was performed in the left eye.

31.10.43. LEFT EYE. The corneal scar was healed. There was a thread-like anterior synechia with a dot-like opacity in the lens at that site.

14.1.44. LEFT EYE. The lens opacity was less marked.

20.1.44. The rabbit was killed and its eyes enucleated.

RABBIT No. 21.

10.10.43. This was a grey doe weighing 3lbs. Fundi were pigmented below the disc and at the lower periphery. The animal was given 0.5 c.c. of nembutal solution and died immediately, apparently from heart failure.

RABBIT No. 24.

This was a black and white doe with rather deeply pigmented fundi.

7.11.43. The animal was anaesthetised with 0.5 c.c. nembutal solution intravenously. The right eye was dislocated and a corneal puncture was performed. The animal was not very deeply anaesthetised and jumped at the critical moment, raising a doubt whether lens or iris were damaged. In the left eye a posterosuperior Graefe puncture was uneventfully performed.

22.11.43. The right eye shewed anterior synechiae. Lens and media were clear. The left eye shewed a small focal reaction. There was no vitreous or lens change.

14.2.44. The eyes were quite quiet and settled.

24.2.44. The animal was killed and its eyes enucleated.

APPENDIX II

Report on Histological Specimens by

JOHN O. OLIVER

Owing to circumstances connected with hostilities only 29 out of the 34 eyes with intra-ocular aluminium or aluminium alloy implants came to sectioning. The two eyes of the control rabbit, No. 20, were also sectioned.

Apart from the fact that the cutting of sections from the eyes of rabbits is an especially difficult technical task, and the lens has generally to be removed, there were other difficulties already indicated in Mr. Savin's text. The sections available for study were, therefore, for the most part rather broken up and difficult to orientate.

The general impression on examining the series of histological specimens is that the clinical findings recorded by Mr. Savin are more important than the histological changes indicated in the sections available for study. In some instances there is no possibility of a complete histological diagnosis from the material available.

In drawing conclusions from the specimens available it is important to remember that the individual rabbits were kept alive for some considerable period (upwards of six months) after the initial experiment had been made so that the earlier pathological changes corresponding to the clinical pictures recorded are no longer visible.

RABBIT 1.

RIGHT EYE. The cornea shows some hyalinisation, but possibly this is merely artefact. The iris (which has been separated from the foreign body mass) shows some thickening and a few small irregular patches of pigmentation. The choroid shows a patch of exudate which is merging with the degenerate retina.

LEFT EYE. The cornea shows a firmly hyalinated zone. The iris shows a few small areas of round celled infiltration and one of these areas contains a foreign body giant cell. The choroid shows some exudate and there is a little degeneration of the retina (less than in the case of the right eye).

RABBIT 4.

RIGHT EYE. The iris is somewhat atrophied and a little pigmented. The sections do not show the presence of retina.

LEFT EYE. The only lesion appears to be some degeneration of the optic nerve.

RABBIT 5.

RIGHT EYE. The cornea shows a healed scar but no pigment is visible. The iris shows well marked atrophy and widespread pigmentation. The retina shows some loss of nerve structure from atrophy and there is thinning of the choroid.

LEFT EYE. These sections are not sufficiently complete for diagnosis but there is a well marked atrophy of the retina.

RABBIT 7.

RIGHT EYE. Very marked retinal atrophy (cystic degeneration in one area).

LEFT EYE. Marked thickening of the cells and some general fibrosis of the iris structure leading to atrophy. Choroido-retinitis well marked with organisation of a thin layer of exudate.

RABBIT 8.

RIGHT EYE. Retinal atrophy almost complete.

LEFT EYE. Some retinal atrophy.

RABBIT 9.

LEFT EYE. Complete disorganisation of the lens structure with marked calcification. This calcification was enforced with ciliary body. There is some fibrosis atrophy of the retina.

RABBIT 10.

RIGHT EYE. Some diffuse pigmentation of the iris. The retina shows an area of round celled infiltration and evidence of general atrophy.

LEFT EYE. Very marked retinal disorganisation with gross exudate present.

RABBIT 11.

RIGHT EYE. Some atrophy of the iris. The greater part of the retina shows complete disorganisation of structure (especially marked at the fundus). Peripherally the changes are less and include some degree of cystic degeneration.

LEFT EYE. Very marked retinal exudate with disorganisation of nervous elements.

RABBIT 12.

RIGHT EYE. Sections are not complete enough for diagnosis but the retinal changes present are seen to be comparatively slight.

LEFT EYE. Unsatisfactory for sectioning.

RABBIT 13.

RIGHT EYE. The iris shows some pigmentation and definite degree of atrophy. The retina shows some exudate throughout but this is very patchy and varies from a microscopical quantity at one point to an exudate equal in thickness to the retina itself in other parts.

LEFT EYE. There is complete disorganisation of the eye. A thick exudate with fibrous bands occupies the anterior chamber. The cornea shows some vascular plaques. The lens of the ciliary body is involved in a large mass of cellular exudate admixed with serous material. There is a vascularisation of the choroid and the retina as such has for the most part disappeared.

RABBIT 14.

RIGHT EYE. The iris shows atrophy and a little pigmentation. There is deposit (clearly metallic fragments and exudate) in the anterior chamber. Retinal changes are not very marked but in one zone there is a thin exudate and two small areas of atrophy.

LEFT EYE. Anterior synechiae. Atrophy of iris. The retina is not well shown in the section.

RABBIT 16.

RIGHT EYE. Anterior synechiae corresponding with an area of partial atrophy in the iris which shows some pigmentation. The retina shows a large area of exudate at the fundus.

LEFT EYE. Cornea incomplete (metallic fragments removed), some atrophy of the iris and well marked choroid exudate.

RABBIT 17.

RIGHT EYE. Cornea shows some vascularisation and "wrinkling" from peripheral contraction. The iris shows atrophy and pigmentation. There is a mass of organised exudate posterior to the iris, which in some of the sections is found to be in association with the remains of the lens. Organised inflammatory exudate is present at the fundus. The retina is not seen.

LEFT EYE. Marked vascularisation of peripheral zone of cornea. Iris atrophic from fibrosis and shows a heavy superficial pigmentation. The retina is incomplete but shows an area of retinitis proliferans.

(Note: This case illustrates particularly well the need for examining all the sections available in every instance since no one section could be found giving the complete story.)

RABBIT 18.

RIGHT EYE. Iris marked atrophy. Retina only minute fragments present.

LEFT EYE. The iris shows atrophic changes, as also the retina. There is considerable débris in the vitreous cavity.

RABBIT 20.

RIGHT EYE. Some vascularisation of the cornea peripherally.

LEFT EYE. No changes seen with the exception of a healed scar of the cornea.

RABBIT 22.

RIGHT EYE. Shows a healed corneal scar. The only other change observed is some superficial pigmentation of the iris and possibly a minor degree of atrophy.

RABBIT 23.

RIGHT EYE. Sections show the whole eye to have been very much shrunken and distorted. The cornea is vascularised and shows a fibrous plaque on the surface at one point. The iris is completely distorted with numerous adhesions of the proliferated epithelial and vascular process. Some inflammatory exudate is present in the margin of the remains of the lens and an organised exudate is also present in the choroid. The retina shows complete degeneration.

ANNOTATION

Tobacco Amblyopia and the Budget.

The Chancellor of the Exchequer, in framing this year's Budget, would seem to have adopted the maxim of Mr. Biggs in Mr. Midshipman Easy who said that duty came before decency. It will be interesting to see whether the increased cost of tobacco will have any influence on the number of cases of tobacco amblyopia in Britain. Doubtless substitutes will be found. We have already heard of husbands who have raided the tea caddy to the despair of the prudent housewife. We have known small boys attempt to smoke blotting paper, and both coltsfoot and hay have been recommended. In passing we may ask whether those who use hay will have to obtain a permit from the War Agricultural Department, sub-section, supply of animal food-stuffs?

The non-smoker sees no virtue in tobacco just as alcohol in any form is anathema to those who have taken the pledge. The writer has smoked tobacco ever since his school days, and was not brought up like the "Infant Bond of Joy," Alfred Pardiggle, who "was pledged never, through life, to use tobacco in any form." Like most other things tobacco can be abused, but used in moderation it never seemed to us to do any harm, and it has a distinctly soothing effect. We find it easier to compose an annotation with a pipe in one's mouth than without it. Burton, *Anatomy of Melancholy*,

Part 2, Section 4, commends its use in moderation, but "as it is commonly abused by most men, who take it as tinkers do ale, 'tis a plague, a mischief, a violent purger of goods, lands, health, hellish, devilish and damned, the ruine and overthrow of body and soul." Here is a sentiment to put in our pipes and smoke.

AMERICAN OPHTHALMOLOGICAL MEETINGS

June, 1947.

AMERICA surely still remains the land of comfort and of plenty and the home of exuberantly kind and hospitable people. I have just had the happy experience of attending the annual meetings of the American Ophthalmological Society and the American Medical Association, two occasions which deserve putting on record,

The 83rd annual meeting of the A.O.S. was held at The Homestead, Hot Springs, Virginia, from June 5—7. Hot Springs, a delightful resort in the heart of the Virginian hills is an ideal location for such a meeting. It consists of a luxury hotel at a rail terminus in the heart of beautiful country where everything pleases except the taste of the warm sulphur spring water—but there are other things aplenty to taste. The Society takes possession of a section of the hotel and works and plays for three days. It is an exclusive Society, limiting itself approximately to 200 members (there are some 8,000 ophthalmologists in the U.S.A.), embracing all that is good in American ophthalmology; practically everyone attends (three consecutive absences from meetings without valid excuse implies expulsion) and all the world brings his wife. The morning is devoted to scientific papers; the afternoon to tennis, golf, swimming or walking, and the evenings to happiness: sleep comes tardily.

The meeting was under the genial presidentship of John W. Burke of Washington and the scientific papers covered a wide field of interest; the subsequent discussions are usually prepared and are more formal than the spontaneous give-and-take we are accustomed to in England. Two cases were reported by Maynard C. Wheeler (N.Y.) of discolouration of the lids from the use of yellow oxide of mercury sustained over many years. Reports of delayed mustard gas keratitis were presented by Walter S. Atkinson (Watertown)—the condition is apparently rarer (or less frequently spotted) than in England. Two cases of an exceptionally rare tumour of the orbit—granular cell myoblastoma—one simple and one malignant, were reported by John H. Dunnington (N.Y.). The occurrence of papilloedema with cerebral oedema in the manifest form of parathyroid deficiency was described by Donald Lyle (Chicago.)

Daniel Kirby (N.Y.) gave an interesting discussion of his technique of dealing intracapsularly with cataract in eyes in which a fluid vitreous was present; he recommended traction with rotation rather than pressure, gripping the lens capsule at 12 o'clock and if necessary rupturing the zonule by stripping it from its attachment to the lens in this region. Wendell Hughes (N.Y.) described his employment of vitallium implants after enucleation; and the first day's session ended with a contribution by Alan C. Woods (Baltimore) on the deterrent effect of promin and promizole (new drugs) on the course of experimental tuberculosis in immune-allergic rabbits.

The second day's session was opened by two papers on the applications of surgical diathermy—in the treatment of angioma of the retina by Philip M. Lewis (Memphis) and of recurring retinal hæmorrhages by F. Verhoeff (Boston): in the latter case one eye had been blinded by recurrent hæmorrhages followed by retinitis proliferans, the other eye was saved (2 years' history) by obliterating on two occasions the segments of the retina wherein hæmorrhages and retinitis proliferans appeared. Gordon M. Bruce (N.Y.) gave an exceptionally interesting paper demonstrating that certain cases of so-called "primary" hypertension were due to a neoplasm (pheochromocytoma) of the adrenal medulla; in such a condition the retinopathy may be the first clinical symptom and the appropriate surgery is therapeutically effective. This is an important new observation. John P. Macnie (N.Y.) presented the results of treatment of aniseikonia over a 5-10 years' interval, claiming some 50% relief from symptoms. Charles E. G. Shannon (Philadelphia) gave a talk on the thyro-pituitary origin of malignant exophthalmos. James N. Greear (Washington) described his technique of orbital reconstruction with buccal mucosa, and the second day's session ended with a discussion of Mikulicz's disease and syndrome by Parker Heath (Detroit).

The final day's session was occupied by a demonstration of difficulties of diagnosis of retinoblastoma by Arthur J. Bedell (Albany), a discussion on agnostic alexia by Harold H. Joy (Syracuse), on diffuse malignant melanomata of the iris by Shaler Richardson (Jacksonville), and on arachnodactylia by Ralph I. Lloyd (Brooklyn). A. B. Reese (N.Y.) gave an interesting talk on glaucoma, introducing the conception of a "base pressure" and a "peak pressure" (corresponding to a diastolic and systolic pressure, the difference being termed the "functional transitory factor" which becomes nil in absolute glaucoma. He pointed out that it is not sufficient to determine a single intra-ocular pressure, but it is necessary to establish whether it represents the lowest, highest or an intermediary point in the pressure curve: the higher the base

pressure, the greater a drainage load any operation must carry. A preliminary report on an investigation to correlate plasma protein fractions with certain types of lenticular changes was presented by A. D. Ruedemann (Cleveland), and an interesting account of his extensive researches into the difficult and exceedingly complicated question of the standardization of the various types of tonometers by Jonas S. Friedenwald (Baltimore). A final paper on pupillometry closed a most informative and friendly meeting.

On the night of Saturday, June 7 a large party of us traveled from Virginia to Atlantic City to celebrate the centenary of the American Medical Association. Most unhappily our journey was marred by the sudden illness and death on Sunday morning of Dr. E. C. Ellett owing to coronary thrombosis. He was a delightful person who took an active part in the proceedings at Hot Springs, and was one of the most charming and beloved of American ophthalmologists. His widow was with us.

Atlantic City is an amazing city on the eastern sea-board which may be partially visualized by piling Brighton on Margate and both on Blackpool; but its mammoth hotels could accommodate the 15,000 delegates to the meeting of the A.M.A.* Monday (June 9) as well as all the spare moments throughout the week, was occupied by the examinations of the American Board of Ophthalmology. This Board gives a post-graduate certification of fitness to practise as an ophthalmic specialist and is a universal criterion throughout the country for full professional recognition and the higher hospital appointments. The examinations (in which I participated) are of a standard corresponding to our D.O.M.S. and have undoubtedly been a most effective means of raising and maintaining the standard of ophthalmology in the United States.

Tuesday was occupied by the Sixteenth Annual Meeting of the Association for Research in Ophthalmology. This is a very valuable Association encouraging and presenting a forum to junior research ophthalmologists and young scientists without a medical degree. Twelve papers were presented and discussed ranging from the bacteriology of seborrhœic blepharitis and conjunctivitis to the transfer of tracer substances through the blood-aqueous barrier.

The meetings of the Section of Ophthalmology of the A.M.A. took place during the next three days under the delightful and efficient chairmanship of that old English friend of ophthalmologists; Dr. Derrick Vail. His Presidential address was on the prestige of ophthalmology wherein, without pulling any punches, he pointed out

* With a corresponding number of wives together with over 3,000 manufacturers and tradesmen who staged a medical exhibition amazing in its interest and comprehensiveness.

the short-comings of ophthalmologists as a class in the United States and how they failed to attain and maintain the position of members of one of the highest specialities of medicine. It is interesting how international are these sins of omission and commission. I gave an address on the nature of the aqueous humour. Thereafter Dr. Verhoeff gave the centennial address—One Hundred Years in American Ophthalmology. The address was most interesting: the story of progress in American ophthalmology during the last century is indeed a proud one, but, characteristically, Verhoeff, was not backward in pointing out what Americans could have done, and had not. The subsequent scientific papers provided unusually interesting and varied fare. Four cases with keratitis presumably due to the virus of lymphogranuloma venereum were described by H. G. Scheie of Philadelphia. A four-year follow-up of 300 glaucomatous eyes was presented by Peter Kronfeld of Chicago: wide-angled and narrow-angled glaucoma is a favourite basis of classification in America, and the latter type was found to carry the better prognosis with regard to vision after control of the tension. An interesting paper on the surgical significance of the ligament of Lockwood in relation to the surgery of the inferior oblique muscles was presented by Walter H. Fink of Minneapolis. The control of experimental ocular infections both external and internal by streptomycin administered systemically and locally was discussed by John C. Bellows and Chester J. Farmer of Chicago: promising results were obtained after infection of the vitreous by streptococci with local treatment by intra-ocular injection but systemic treatment did not prevent infection; corneal infections by *B. pyocyaneus* were aborted by local treatment. Unilateral syphilitic optic atrophy was discussed by Walter L. Bruetsch of Indianapolis, and the value of sodium sulphacetamide in ophthalmology by Leo Mayer of St. Louis. Conservative treatment of congenital impatency of the nasolacrimal duct was advocated by Edwin L. Kendig and Du Pont Guerry of Richmond; and late operation (after $2\frac{1}{2}$ years) in the surgical treatment of congenital cataract by W. C. Owens and W. P. Hughes of Baltimore. Three interesting papers related to the war experiences of some of the younger men—on intra-ocular foreign bodies by G. M. Haik of New Orleans, on military ophthalmology by Don Marshall, and on plastic reconstruction of the lids by S. M. Dupertuis of Pittsburg, in the subsequent discussions of which several of our old friends whom we knew in the European theatre of war participated.

All these, with official receptions and functions, dimmers (public and private), and good fellowship abounding, constituted a memorable week indeed.

STEWART DUKE-ELDER.

BOOK NOTICE

Clinical Methods of Neuro-Ophthalmologic Examination. Kestenbaum. \$6.75. Greene and Stratton, New York, 1946.

In the introduction to this volume, the author states that its object is "to present a review of the clinical methods of ophthalmologic examination that may be useful in neurologic differential diagnosis." Anatomical data and theoretical experiments are therefore recorded only in so far as they are required for the understanding of the methods described. The book is thus essentially a practical one and is not intended to supersede well-known works on neuro-ophthalmology. Since many ophthalmologists are often not concerned with methods of neurological localisation, and many neurologists are not concerned with ophthalmological methods, the science with which this book deals has, up to date, suffered neglect. Such is no longer the case, and anyone who has read *Neuro-Ophthalmologic Examination* will have all the information needed to enable him to elicit and interpret the eye signs and symptoms in a case of nervous disease. The author is at pains to point out, however, that such signs and symptoms are only a contribution to the final diagnosis in the same way as are radiological findings, and that the last word must always rest with the neurologist.

As an instance of the thoroughness with which the subject is tackled, it may be mentioned that no fewer than five different methods of non-instrumental perimetry are described, and that in describing instrumental perimetry four common errors of procedure are mentioned, in the hope that by elimination of them the perimetrist will "measure the real visual field, and not the visual acuity, the degree of attention or the size of the palpebral aperture."

Since the author is a well-known authority on his subject, his book contains much that is new, and the result of his own thoughts and investigations. The description of the positions of retinal haemorrhages is a case in point. The disc is regarded as a clock face, and the prefixes A, V and C designate artery, vein and small vessel respectively. A vein crossing the disc at 2 o'clock would be designated V2; the branches into which it breaks up are also numbered in a clockwise direction. V2.2.2. would, therefore, be the right-hand branch of the right-hand primary bifurcation of a vein which crossed the disc at 2 o'clock.

The value of the text is enhanced by the inclusion of over 20 tables, which are useful for hasty reference. One of these deals with the 8 different types of chiasmal syndromes, and correlates them with their most common causes; another gives 13 different signs which may aid in localisation of the cause of hemianopia.

Kestenbaum is well known for his work on nystagmus, and as would be expected, his chapter on this difficult subject is full of useful information which is conveniently summarised in tabular form.

Disturbances of ocular movements are also very fully gone into, under the headings of Eye muscle palsy, Gaze movement and Gaze palsy. Various methods are described, but no mention is made of the Hess Screen, possibly because the author is not satisfied with it.

In dealing with the pathology of the optic nerve, the author has much valuable information to give, but he does not mention the absence of papilloedema in purely pontine growths, and the frequent association of a sub-tentorial tumour with the presence of a macular fan.

The characteristics of "neuro-ophthalmologic examination" appear to be:— clarity of expression; simplicity of technique for tests, and the incorporation of much information of practical value which is not obtainable elsewhere. The practical nature of much of the text makes this book a difficult one to review, particularly by armchair reading. One has the feeling all the time that it should be taken into the Clinic, where a far better appraisal of its value could be made by using it as an aid to the investigation of patients.

Dr. Kestenbaum is to be congratulated on having produced a volume of outstanding value.

CORRESPONDENCE

CONJUNCTIVITIS IN EXPOSURE TO DIMETHYL-SULPHATE

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—In your latest issue of June (p.373), H. J. Stern describes a case of conjunctivitis due to exposure to vapours of dimethyl-sulphate (abbrev. D.M.S.). He also cites papers of Weber and of different authors, all published early this century. With respect to the rarity of poisoning with D.M.S., it would be of interest to recollect the cases seen at Budapest in 1934. At one of our chemical works, a 15lb. glass container filled with D.M.S. was broken by a labourer. One out of the three injured came under care of J. Petres, the other two were seen by the laryngologist G. Boskowitz and myself. (Case record in the Rep. on the 4th General Meeting of Hungarian Physicians 1934). The most severe injuries which ended fatally under symptoms of lung-oedema on the fourth day occurred in the girl who had caused the accident. Thirteen hours

after the injury, tracheotomy had to be performed on account of sudden onset of glottis-oedema; this was followed by a membranous laryngo-tracheo-bronchitis. This massive poisoning resembles that with chlorine, the action of both substances having much in common. With the other patients, there was only some irritation of the respiratory tract. Eye symptoms set in at the very moment of contact with the fumes in all three cases and corresponded to those described by Stern, except that the cornea showed multiple punctate erosions and in the superficial layers round-shaped faint infiltrations which have proved most resistant to therapeutic procedures. A remarkable symptom consisted in corneal hypaesthesia, D.M.S. being a so-called anaestheticum dolorosum. Several weeks passed before the eyes recovered. For first aid we performed lavation with freshly prepared sodium bicarbonate solution. Later, alkaline ointment which had been used against gas-injuries during the first World War, was applied. (Rp. natr. borac. 0,1, natr. bicarb. 0,2, adip. Lanae, aqu. dist. aa. 1,0, vasel. flav. 8,0.)

Yours faithfully,

CORNELIUS RAÁB, M.D.

BUDAPEST, V. GEZAU. 3.

June 25, 1947

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs.—In one of your last issues (June) Mr. H. J. Stern gave an account of eye-injury due to dimethyl-sulphate fumes. May I call your attention to my paper published in the *Amer. Jl. of Ophthal.* (Vol. XX, No. 7, 1937) where the question is fully reviewed. The typical ocular findings are following latency (hydrolysis), corneal lesion varying from mild roughening to extensive erosion, accompanied by photophobia, lacrimation, etc. For treatment I recommend alkaline cream, as possibly the local changes are due to sulphuric acid. On the other hand, methyl alcohol, the other split-product may cause similar erosions on the cornea. Cocaine seems to be contraindicated owing to its undesirable effect on epithelium. Irritating atmosphere should be avoided in after-care. Homatropine is of benefit against sphincter spasm.

I am, Sir,

Yours very sincerely,

STEPHEN DE GRÓSZ, M.D.

Associate-Professor, Eye-Hospital,
University Medical School,
Budapest.

Budapest, Hungary.

POSITION AND MOVEMENTS OF THE EYE IN
OCULOMOTOR PARALYSIS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS.—In their note on the position of the eye in third nerve palsy, Mr. Wolff and Surgeon-Commander Heffernan raise several interesting questions. They state quite correctly that the usual description given of the position of the affected eye is "out and somewhat down or simply down and out." The illustrations, however, published in several text books show usually a relative depression of the cornea of the affected eye, which appears to be due to the photograph having been taken with the unaffected eye looking slightly upwards. The text-books are unhelpful in that the descriptions are vague and inexplicit and, an important point, do not state whether the paralysis was complete or not or in what direction the patient was looking when the picture was taken. In one text-book an illustration of bilateral IIIrd paralysis shows both corneae elevated rather than depressed. This obscurity in the text-books arises largely from the habit of copying statements from one book to another without critical examination. I found the following in two text-books:—

"Finally the eye is deviated outward and a little downward because the two muscles that are not parietic, the external rectus and the superior oblique, draw the eyeball in this direction."

"... the eyeball, which is deflected strongly outwards and somewhat down, because the two muscles not paralysed—the external rectus and the superior oblique—draw it in this direction."

It is not likely that in complete IIIrd palsy any apparent downward direction of the affected eye could be produced by the action of the superior oblique and the above statements must be regarded as incorrect.

The second question is:—"What are the movements of the affected eye?"

Berry (1889) explains clearly that the amount of depression possible depends on the position of the eye. If the paralysis is incomplete so that a little power remains in the medial rectus the superior oblique can come into action as a depressor. In such cases a little power might also remain in the inferior rectus. It is interesting to note that in the case described the affected eye could be brought to the midline, presumably owing to relaxation of the left lateral rectus, and yet the patient was unable to turn the cornea downwards. One would be glad to have the authors' comment on this feature of their case.

The late W. G. Sym used to pose a test question to examination candidates:—"How does the eye move in oculomotor paralysis when the patient is asked to look down?" The correct answer was "Nasal rotation of the cornea only". I have myself never seen depression of the cornea in complete IIIrd paralysis.

The third interesting point is the absence of any affection of the optic nerve in the case recorded. It is noteworthy that in cases of a blow to the temple in which the optic nerve is damaged the motility of the eye is unimpaired and where a traumatic paralysis occurs the optic nerve escapes.

Yours faithfully,

H. M. TRAQUAIR.

EDINBURGH.
July 8, 1947.

COLOUR VISION IN THE CONSULTING ROOM

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs.—I was pleased to see the letter from John Grieve regarding my article on colour vision, and am glad to give the explanation he requires.

The article, "Colour Vision in the Consulting Room," was a very brief résumé of some work I had done which was not published in full owing to the paper shortage, and naturally any detailed descriptions were omitted.

I made no claim that the figure of 5.5 per cent. for the gross incidence of colour defectives was anything other than the actual percentage of the subjects I had examined. These men were not chosen at random from the male population and any generalisation would have been misleading.

I consider it would have been a waste of space to describe the correct technique for using the Ishihara plates in an ophthalmological journal, and likewise considered a detailed description of the construction of the various lanterns, some of which were made from black-out shades and coffee tins, entirely irrelevant: the number and size of the apertures and the use of a rheostat appeared to be the only data necessary. The filters used approximated to those recommended by the International Committee for Aerial Navigation.

No laboratory methods could be applied to my tests, hence the title I used, and variations in colour temperature were ignored.

I hope that the résumé had made it clear that single and multi-light lanterns functioned differently, the first emphasising successive contrast, the second simultaneous contrast, and/or successive, if, for the sake of argument, one supposes that macular fixation allows only one object to be seen at a time, a point which it is not my intention to discuss.

Yours truly,

FRANK R. NEUBERT.

HAUTERIVE,
THE QUEEN'S ROAD,
GUERNSEY.
July 12, 1947.

VISUAL DEFECTS IN ARMY RECRUITS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—R.A.M.C. Ophthalmologists have recently completed a survey of visual defects amongst the recruit intake in the United Kingdom.

Some figures were obtained relative to men whose uncorrected vision in one eye was 6/18 and below.

During the survey of a recruit population of 13,705, some 1,099 men were found to have monocular vision of 6/18 and below.

ANALYSIS.

Cause.	Percentage of Partial Amblyopes.	Percentage Recruit Intake.
High Refractive Error	39.2%	3.1%
Strabismus	32.7%	2.6%
Trauma	10.7%	0.9%
Inflammation... ..	6.2%	0.5%
Degeneration... ..	6.1%	0.5%
Congenital Defect ...	1.6%	0.1%
Cause Undetermined	3.5%	0.3%
		= 8%

Comparable figures were published in the Archives of Ophthalmology (1944, Vol. XXXI, May, p. 399-402) by Theodore, Johnson Mills and Bonser. They examined 10,532 men for visual defect out of a recruit population of 190,012.

ANALYSIS.

Cause	Percentage of Partial Amblyopes.	Percentage Recruit Intake.
High Refractive Error	24.9%	1.4%
Strabismus	24.2%	1.3%
Trauma	18.1%	1.0%
Inflammation... ..	1.9%	0.1%
Degeneration... ..	1.0%	0.06%
Congenital Defect ...	2.3%	0.13%
Cause Undetermined	27.6%	1.53%
		= 5.5%

Periodic "sample" surveys have been taken by the Ministry of Labour and National Service Candidates rejected for visual defects:—

1939—1941	1.08%	of all candidates rejected for visual defect.
1942	1.13%	" " " "
1943—1944	0.7%	" " " "

The Ministry of Education pamphlets give estimates of 0.02-0.03% for blind children, and 0.1% for partially sighted children for all registered pupils. Such children will ultimately constitute a large group amongst the National Service Board rejects.

The present Visual Standard applied in the Army may be summarized as follows:—

- Standard One* Unaided vision to 6/6 in the one eye and 6/9 in the other.
- Standard Two* Unaided vision is either 6/12 in both eyes or Right eye 6/6 and Left eye 6/36.
- Standard Three* Vision correctable to Standard Two.
- Standard Four* Vision correctable to 6/12 in the one eye, and 6/36 in the other. Right or Left eye can be the "Master" eye.
- Standard Five* Vision correctable to 6/24 in both eyes.
- Standard Six* Vision correctable to 6/12 in the one eye and in the other not less than 6/60. There are limitations to the degrees of myopia or hypermetropia permissible.
- Standard Seven* Rejects or especially retained low category serving soldiers.

A recent analysis of the Ministry of Labour showed that the intake figures for men in Visual Standard Six were 0.6% of the total.

The British figures of 8% and American 5.5% would appear to cover most of the defects from Standard Two-Standard Six.

It is felt that the results of the investigation may be of interest to school medical authorities, industrial ophthalmologists and the future National Health Ophthalmic services.

Yours faithfully,

G. C. DANSEY-BROWNING,

Lieut.-Colonel, R.A.M.C.

Adviser in Ophthalmology.

Q. A. MILITARY HOSPITAL,
MILLBANK, S.W.1.

June 6, 1947.

NOTES

National Society for
the Prevention
of Blindness

CONRAD BERENS, M.D., of New York City, was elected vice-president of the National Society for the Prevention of Blindness at the semi-annual meeting of the Board of Directors, on June 6, 1947. Eugene M. Geddes, also of New York, was elected treasurer, succeeding George C. Clark.

Re-elected were the Society's president, Mason H. Bigelow, of New York; two other vice-presidents, Preston S. Millar, of New York, and Russell Tyson, of Chicago; and, as secretary, Regina E. Schneider, of New York.

Sociedad de
Oftalmología del Litoral

The Officers of the Sociedad de Oftalmología del Litoral for the session 1947-1948 are as follows:
President: Dr. Enrique V. Bertotto. *Vice-President*: Dr. Arturo Reca. *Secretary*: Dr. Juan Manual Vila Ortiz. *Treasurer*: Dr. Roberto Giqueaux. *Council*: Drs.: Luis A. Gallo, Fernando Miquelarena, Benito Bianco y Cayetano D'Eramo. The new address of the Society is (Italia 663, local del Círculo Médico, Rosario, Argentina.)

* * * *

The
Ophthalmological
Society of
New Zealand (B.M.A.)

IT is a matter for congratulation to all concerned that, in February last year, at the Biennial meeting of the B.M.A. held in Auckland, New Zealand, the Ophthalmological Society of New Zealand was formally constituted.

The first annual conference was held in Auckland in February, 1947, the second will be held in Dunedin from February 3-6, 1948. The Patron of the Society is Sir H. Lindo Ferguson, C.M.G., the sole remaining original member of the Ophthalmological Society of the United Kingdom. The following officers were elected in 1946.
President:—W. A. Fairclough; *Vice-President*:—G. W. Harty; *Executive*:—H. Coverdale, E. L. Marchant, R. P. Wilson; *Hon. Secretary Treasurer*:—W. J. Hope Robertson; *Hon. Secretary of Conference*:—C. A. Pittar. The same officers with the addition of Dr. L. A. Lewis as an extra Vice-President will serve for the current year.

The first conference was held at the Auckland Hospital: the programme was varied and interesting.

We extend a most cordial welcome to the New Zealand Ophthalmological Society and hope that it may be possible in future years for us to publish a brief survey of each annual conference.

* * * *

American
Ophthalmological
Society: Officers

THE officers elected at the recent annual meeting of the American Ophthalmological Society are as follows:—

President:—Dr. Henry C. Haden; *Vice-President*:—Dr. Bernard Samuels; *Editor*:—Dr. Wilfred E. Fry; *Secretary-Treasurer*:—Dr. Walter S. Atkinson.

* * * *

Courses in
Illuminating
Engineering.

IN response to the demand for courses in Illuminating Engineering, two such courses have been arranged to take place in London during the period September, 1947 to May, 1948, one at the Northampton Polytechnic on Wednesday afternoons and evenings, the other at the Borough Polytechnic on Thursday afternoons and evenings.

The courses will prepare candidates for the City and Guilds of London Institute intermediate examination in Illuminating Engineering.

A course has also been arranged at the Central Technical College, Birmingham, to commence in September this year and other courses are in prospect at Cardiff, Leeds, Liverpool and Manchester, further details of which will be available from the Illuminating Engineering Society, 32, Victoria Street, London, S.W.1, in due course.

* * * *

As Foreign Guest of Honor at the Centennial Meeting of the American Medical Association in June, 1947, at Atlantic City, Sir Stewart Duke-Elder was made an Honorary Fellow and presented with the Ophthalmic Research Medal of the Association.

* * * *

Transactions of the
Ophthalmological
Society of the
United Kingdom.

BACK numbers of the Transactions may be obtained through Headley Brothers, The Invicta Press, Ashford, Kent, at the charge of £1 to members of the Society and £2 to non-members.

* * * *

Corrigenda

UNFORTUNATELY authors' corrections of the paper by Professor E. Th. Levkoieva were not received till June 15. The following corrections should be noted: p. 339, thirteen lines from the bottom of the page should read "in 1942, among 364 enucleated traumatised eyes." Two lines lower down "In 412" eyes enucleated should read "In 42" eyes. Page 342—end of first paragraph "H. K. Müller, in 1945 in his book," should read, "H. K. Müller, in 1945, in his paper printed in the book." (Editor, R. Thiel). Page 345—end of first paragraph should read "The characteristic triangle . . . was seen by us in the majority of all examined cases of Kuhnt's operation in small perforating wounds when regeneration is obvious." Page 348—3 lines below illustration for "diascleral" read "episcleral," Page 353—Last paragraph but two (Preobrajenskaja, M.N.) for G.N. Page 358—Fig. 9, legend should read "Interstitial Argyrophil substance stained by Snezarev's method."

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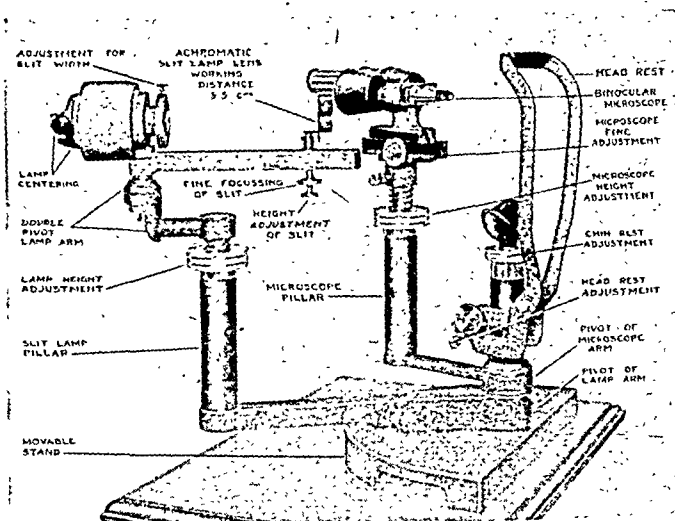
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THE BRITISH JOURNAL OF OPHTHALMOLOGY

SEPTEMBER, 1947

COMMUNICATIONS

DISTRIBUTION OF PENICILLIN IN THE EYE AFTER SUBCONJUNCTIVAL INJECTION*

BY

ARNOLD SORSBY *and* J. UNGAR

LONDON

ADEQUATE intra-ocular concentrations of penicillin can be reached by both systematic administration of massive doses (Struble and Bellows, 1944; Town, Frisbe and Wisda, 1946; Town and Hunt, 1946 and Sorsby and Ungar, 1946) and by the application of highly concentrated ointments and subconjunctival injections (Sorsby and Ungar, 1946. In a previous study we indicated that the highest and most persistent intra-ocular concentration is obtained by means of subconjunctival injection especially when combined with adrenalin; the first of these findings has been confirmed and extended by Andrews (1947) and the second by the data recorded in the present communication.

The technique employed is that recorded in our earlier study.

* Received for publication, July 7, 1947.

Cocaine analgesia is used. Each of our values represents the average of two findings, which generally showed a close approximation. Where adrenalin was used, a solution 1:1,000 was the solvent employed instead of water.

Experimental data

1. *Preliminary experiments on the effect of adrenalin.*—Table I shows the intra-ocular levels reached on the subconjunctival injection of 20,000 units of penicillin with and without adrenalin. Though there are slight discrepancies the following conclusions seem valid :—

(1) One hour after injection the level of penicillin in the various intra-ocular tissues is generally rather higher where adrenalin had been used.

(2) After 3 hours the difference is more distinct. Where adrenalin had not been used penicillin tends to disappear from the eye. In contrast substantial intra-ocular levels are present where adrenalin was employed.

(3) Both these findings are reflected in the uninjected eye: where adrenalin has been used the intra-ocular levels are rather higher and definitely more persistent.

It would therefore seem that adrenalin acts by preventing the rapid elimination of the penicillin from its subconjunctival depot.

2. *Effect of dose on intra-ocular levels.*—When 50,000 units of penicillin are injected subconjunctivally the intra-ocular levels reached are generally considerably more than 2½ times those obtained with 20,000 units, as can be seen from a comparison of the corresponding data in Table I. Moreover it would seem that the levels reached are more persistent.

The following summary table brings out some of the salient features.

Subconjunctival injection	Level in										
	Aqueous		Vitreous		Cornea		Anterior uvea		Posterior uvea		
	At	1 hr.	3 hrs	1 hr.	3 hrs.	1 h.	3 hrs.	1 hr.	3 hrs.	1 hr.	3 hrs.
20,000 units ...	10.0	0.06	0.5	Trace	>30	0.8	>6.1	0.375	11.5	0.38	
50,000 units ...	17.0	3.0	5.0	1.50	76	18.75	70.0	8.75	140	11.25	

This disproportion is emphasised when the values obtained for injection with adrenalin are examined, as can be seen from the following summary table.

Subconjunctival injection with adrenalin	Level in									
	Aqueous		Vitreous		Cornea		Anterior uvea		Posterior uvea	
At	1 hr.	3 hrs.	1 hr.	3 hrs.	1 hr.	3 hrs.	1 hr.	3 hrs.	1 hr.	3 hrs.
20,000 units ...	1.0	0	2.0	0.125	>80	>10	>14	>3	>32	>8
50,000 units ...	>32.0	>20	17	0.5	>1,440	450	775	60	>750	20

3. *Effect of adrenalin on intra-ocular levels.*—Comparison of the data in Tables II and III shows the marked effect of adrenalin in raising the intra-ocular levels of penicillin. The effect is apparent within a quarter of an hour and would appear to be maximal at 2—3 hours, declining after that, so that at 6 hours the adrenalin values are generally of the order of twice those obtained without adrenalin.

The following summary table and figs. 1—4 bring out some salient features.

Penicillin levels

At	$\frac{1}{4}$ hr.	1 hr.	2 hrs.	3 hrs.	6 hrs.
Aqueous					
without adrenalin ...	24	17	9	3	0.56
with adrenalin ...	>32	>32	>32	>20	1.25
Vitreous					
without adrenalin ...	7	5	1.5	1.5	0.06
with adrenalin ...	>17	17	5.0	0.5	0.03
Cornea					
without adrenalin ...	950	76	25.0	18.75	1.5
with adrenalin ...	>860	>1440	925	450	2.5
Anterior uvea					
without adrenalin ...	160	70	1.25	8.75	0.34
with adrenalin ...	>290	>775	510	60	0.6
Posterior uvea					
without adrenalin ...	200	140	9.5	11.25	1.12
with adrenalin ...	>550	>750	230	20	0.6

TABLE I

Comparison of the Distribution of Penicillin in Ocular Tissues after Subconjunctival Injection of 20,000 units Pure Penicillin with and without Adrenalin.

	Penicillin levels in units per c.c. of fluid or per gm. of tissue			
	1 hour after injection		3 hours after injection	
	Without adrenalin	With adrenalin	Without adrenalin	With adrenalin
INJECTED EYE				
Aqueous	10	1.0	0.06	0
Vitreous	0.5	2.0	Trace	0.125
Cornea	>30	>80	0.8	>10
Lens	0.25	1.0	0	0.06
Anterior sclera ...	>7.5	>12	0.95	>4
Posterior sclera ...	>12	>12	1.25	>4
Anterior uvea ...	>6.1	>14	0.375	>3
Posterior uvea ...	11.5	>32	0.38	>8
Optic Nerve... ..	1.0	1.0	0	0.5
Extra-ocular muscles	>18	12	0.75	>4
BLOOD	4.5	8	0	1
UNINJECTED EYE				
Aqueous	2.125	1.0	0	0
Vitreous	0.03	0.25	0	0.125
Cornea	0.487	1.25	0.63	3.5
Lens	0.03	0		0.125
Anterior sclera ...	1.32	3.0	0.5	3.5
Posterior sclera	0.84	1.5	1.25	>4
Anterior uvea ...	0.3	0.25	0.09	0.6
Posterior uvea ...	0.15	4	0.18	1.5
Optic Nerve... ..	0.125	0.5	0	0
Extra-ocular muscles	0.37	4	Trace	1.2

TABLE II

Distribution of Penicillin in Ocular Tissues after Subconjunctival Injection of 50,000 units Pure Penicillin in 0.5 of Distilled Water

		Penicillin levels in units per c. c. of fluid or per gm. of tissue						
Hours after injection :		$\frac{1}{4}$ hr.	$\frac{1}{2}$ hr.	1 hr.	2 hrs.	3 hrs.	4 hrs.	6 hrs
INJECTED EYE								
Aqueous	24	19	17	9	3	0.75	0.56	
Vitreous	7	2.5	5	1.5	1.5	0.12	0.06	
Cornea	950	97	76	25	18.75	4.87	1.5	
Anterior sclera ...	>660	380	120	11	16	7.25	2	
Posterior sclera ...	580	450	153	30	15.4	8.5	2	
Anterior uvea ...	160	60	70	1.25	8.75	4.2	0.34	
Posterior uvea ...	200	110	140	9.5	11.25	Trace	1.12	
Extra-ocular muscles	310	140	140	23.75	17.5	9.5	1	
BLOOD		16	16	8	0.35	0	0	0
UNINJECTED EYE								
Aqueous	2	2	1	0.5	0.25	0	0	
Vitreous	0.5	0.12	0.25	0.12	0	0	0	
Cornea	4	4	4	1	1.5	0	0	
Anterior sclera ...	1	1	1	0.5	0.6	0.3	0.3	
Posterior sclera ...	3	3	1.5	1.5	1.4	0.8	0.4	
Anterior uvea ...	2.5	2.5	1.25	0	0	0	0	
Posterior uvea ...	5	2.5	2.5	0	0.1	0	0	
Extra-ocular muscles	6	6	4	0	0	0	0	

Fig. 5 shows graphically the levels reached with adrenalin.

4. *Effect of adrenalin on blood-level.*—From Table II it would appear that where adrenalin is not used, the maximum blood level is reached within half an hour, after which it rapidly declines

to zero by the end of the second hour. Where adrenalin is used (Table III), lower but more persistent levels are reached. The following summary table and the revelant graphs in Figs. 1 and 3 bring out the salient features.

TABLE III

Distribution of Penicillin in Ocular Tissues after Subconjunctival Injection of 50,000 units Pure Penicillin in 0.5 ml. of Adrenalin 1:1,000

Hours after injection:	Penicillin levels in units per c.c. of fluid or per gm. of tissue						
	$\frac{1}{2}$ hr.	$\frac{1}{2}$ hr.	1 hr.	2 hrs.	3 hrs.	4 hrs.	6 hrs.
INJECTED EYE							
Aqueous	>32	>17	>32	>32	>20	20	1.25
Vitreous	>17	>17	17	5	0.5	13	0.03
Cornea	>860	>200	>1,440	925	450	65	2.5
Anterior sclera ...	>290	>285	>450	295	185	50	4.0
Posterior sclera ...	>400	>240	>520	450	96	19	1.25
Anterior uvea ...	>290	>220	>775	510	60	13.5	0.6
Posterior uvea ...	>550	>200	>750	230	20	6.5	0.6
Extra-ocular muscles	>2,200	1,400	1,200	560	92.5	50	9.0
BLOOD							
	>2	>3	3	1.5	0.75	0.12	0.12
UNINJECTED EYE							
Aqueous	3.25	0.37	0.25	0.5	0.19	0.12	0.3
Vitreous	0.06	0.3	0.06	0.06	0.25	0	0.05
Cornea	2.75	3.75	1.25	0.625	1.2	1.75	0.12
Anterior sclera ...	3.50	1.75	4.4	2.1	0.58	0.75	0.65
Posterior sclera ...	3.8	2.8	3.75	2.1	0.85	0.6	0.7
Anterior uvea ...	5.7	2.25	5.65	0.8	0.33	1.5	0.1
Posterior uvea ...	9.0	3.50	1.25	0.62	1.4	0	0.3
Extra-ocular muscles	9.0	2.65	3.0	3.5	0	0.6	0.25

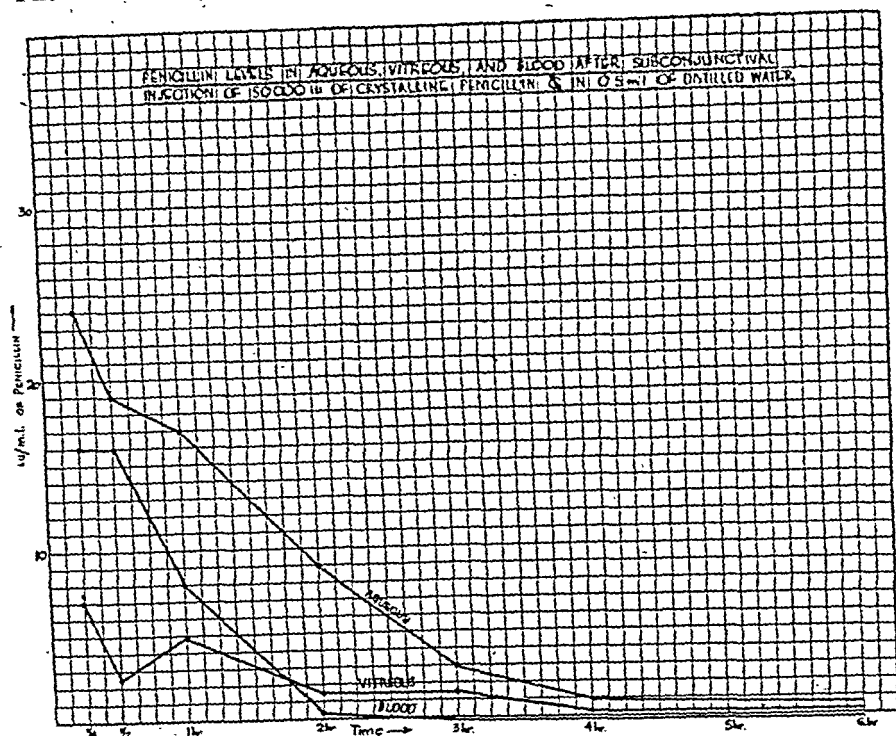


FIG. 1.

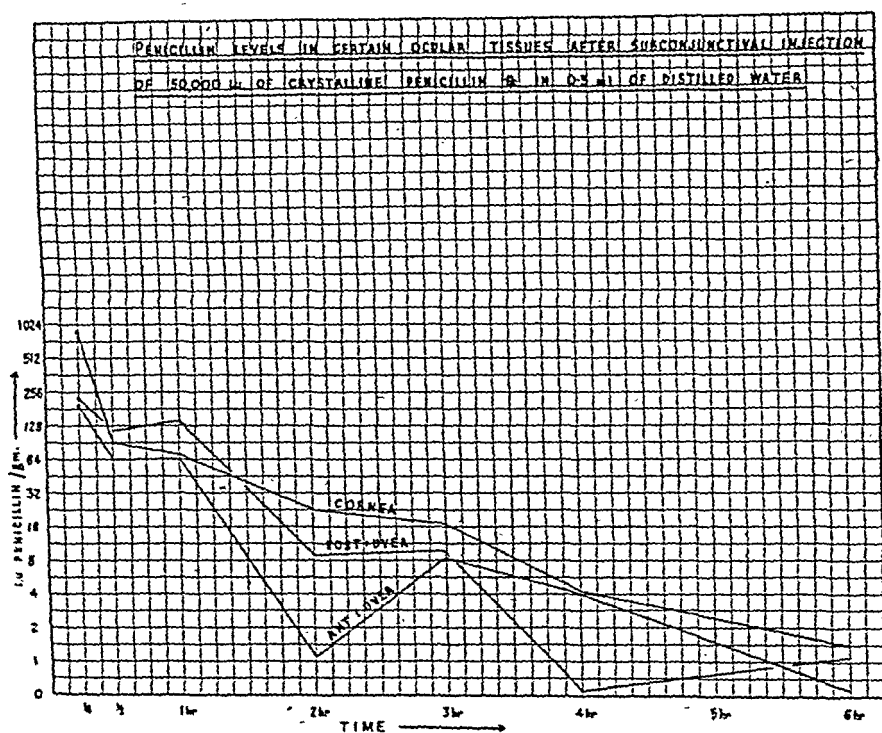


FIG. 2.

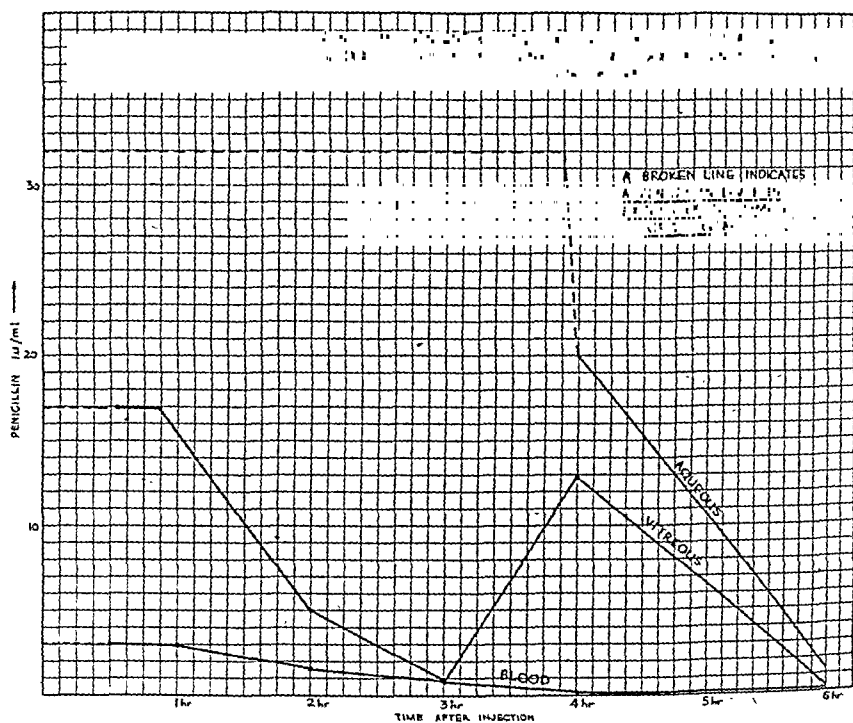


FIG. 3.

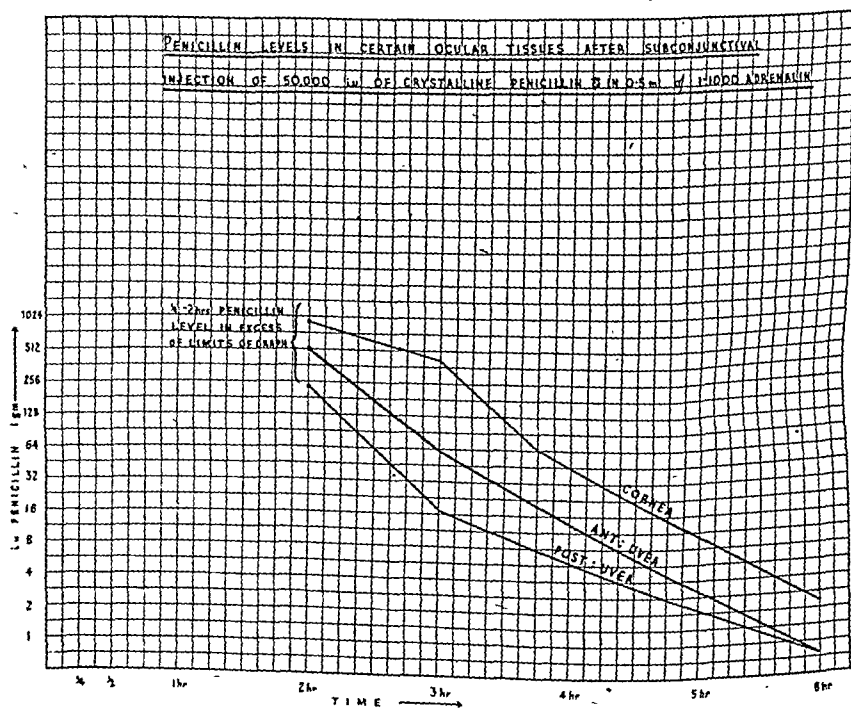


FIG. 4.

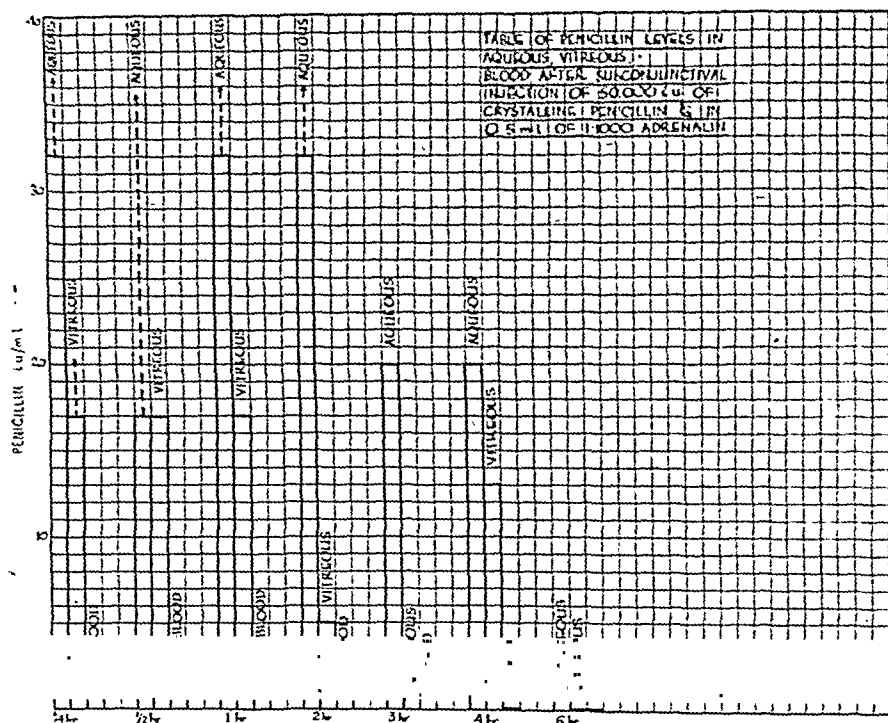


FIG. 5.

Subconjunctival injections of 50,000 units of penicillin	Blood-levels at				
	$\frac{1}{4}$ hr.	1 hr.	2 hrs.	3 hrs.	6 hrs
Without adrenalin	16	8	0.35	0	0
With adrenalin	>2	3	1.5	0.75	0.12

5. *Effect of adrenalin on the levels reached in the uninjected eye.*—As can be seen from Tables II and III there is a considerable intra-ocular level of penicillin in the uninjected eye after a massive dose subconjunctivally in the fellow eye. Comparison between the levels reached on the injection of penicillin with and without adrenalin is difficult, as these levels are generally low and rather bizarre. It is, however, clear that intra-ocular levels tend to persist longer when adrenalin is used. When injections are made without adrenalin all the ocular tissues except the sclera are free from penicillin at 4 and 6 hours, whilst fairly substantial levels are still present where adrenalin has been used.

6. *Observations in man.*—When eyes of patients came to excision the opportunity was taken to inject penicillin subconjunctivally at variable intervals before excision. The level of penicillin in the aqueous and vitreous was determined by assay of some aspirated fluid. The following table gives the results obtained.

Level of penicillin in the aqueous and vitreous of human eye after subconjunctival injection of 50,000 units of penicillin in 0.5 c.c. of 2 per cent. novocaine

Patient's Age	Time injected before enucleation	Concentration in		Clinical Condition	Remarks
		Aqueous	Vitreous		
74	$\frac{1}{2}$ hour	16	0	Old irido cyclitis	—
12	1 hour	2	0.25	Old intra-ocular foreign body	—
5	1 hour	4	0.125	Old irido-cyclitis	—
27	2 hours	32	0.5	Intra-ocular foreign body. Panophthalmitis	—
55	4 hours	0.5	16	Old injury. Iris bombe	Fluid vitreous

These limited results are consistent with the experimental findings, in so far as they show that adequate therapeutic levels can be reached in both the aqueous and vitreous, and much more readily in the first than in the second.

Discussion

1. *Comparison with previous data.*—Andrews' data, like our preliminary results, have shown that high intra-ocular levels are reached by subconjunctival injection of 50,000 units of penicillin. Though his actual values are consistently lower, the findings recorded by Andrews agree closely with our own. The significant differences in the two studies are that whilst Andrews found the concentration in the vitreous almost uniformly negligible, the concentrations we obtained were therapeutically significant. In our present investigation we ignored the lens, as in preliminary experiments we found the concentration in the lens consistently negligible. Andrews' readings showed rather more positive values.

2. *Comparison of levels reached by subconjunctival injection with those obtained by ointments and systemic administration.*—The only data available are those recorded in our previous study.

It may be recalled that ointments in concentrations of up to 40,000 units/gm. do not maintain any adequate intra-ocular levels after 2½ hours. To a lesser extent this also applies to massive systemic administration. The following summary table brings out salient comparative data on the use of subconjunctival injections, ointment and systemic administration of penicillin.

Concentration at 2 hours after application

	Aqueous	Vitreous	Cornea	Sclera	Uvea
Subconjunctival injections 50,000 units					
Without adrenalin ...	9	1.5	25	20.5	5.37
With adrenalin ...	>32	5.0	925	372.5	370
0.1 gm. of ointment 50,000 U/gm.	2.75	4	0.75	1	0.3
Intravenous injections * 25,000 units	0.37	1	0.5	2	2.05

* After 2½ hours.

Though the values recorded for intra-ocular levels after the application of ointment and after systemic administration are based on few observations, the overwhelming advantages of subconjunctival injections are beyond question.

3. *Factors in the maintenance of intra-ocular levels of concentration.*—The level of concentration of penicillin intra-ocularly depends on the amount of penicillin that reaches the eye, on the amount eliminated either by excretion or destruction, and on the rate at which these processes take place. Of the many factors involved only the dose of penicillin and the addition of adrenalin to the penicillin have so far been isolated. For adequate control of intra-ocular levels an intensive study of other relevant factors is required.

4. *Clinical applications.*—From the data recorded it is clear that an injection of 50,000 units is preferable to one of 20,000 for two reasons: in the first place it gives considerably and disproportionately higher levels of intra-ocular concentration; and secondly, these levels are maintained for a longer period. It is also clear that the solvent used is a matter of some importance. Normal saline is excluded as any substantial amount of penicillin dissolved in such a solution makes it hypertonic (Ungar and Denston, 1946). The advantages of adrenalin have already been indicated. For repeated injections, particularly in an inflamed eye, 2 per cent. novocaine as a solvent has considerable advantages, and there

would seem to be no reason why equal quantities of adrenalin and novocaine should not be used as a solvent for routine purposes. As for the frequency of application, results in the rabbit suggest that when 50,000 units are used injections should be given at intervals of 6 hours.

Summary

1. Substantial concentrations of penicillin in the ocular tissues, many times the usual therapeutic level, can be obtained by the subconjunctival injection of crystalline penicillin in a dose of 50,000 units. Adequate levels persist for 6 hours.

2. The concentrations are distinctly higher if adrenalin 1:1,000 is used as the solvent for the penicillin.

3. Observations on 5 human eyes support the findings obtained experimentally.

Our thanks are due to Dr. H. M. Walker of Glaxo Laboratories Ltd., for facilities for the work recorded and to Mr. B. Helliwell for his painstaking technical assistance.

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LOCAL PENICILLIN THERAPY OF HYPOPYON FORMATION: WITH SPECIAL REFERENCE TO THE USE OF SUBCONJUNCTIVAL INJECTION*

BY

ARNOLD SORSBY and HOWARD REED

LONDON

INDICATIONS for the use of penicillin and optimal methods for its employment locally have still to be determined. Drops require to be instilled at frequent intervals; lamellae have not proved satisfactory and ointments have presented difficulties as to the best base to be employed. For hypopyon ulcer Juler and Young (1945) have found penicillin effective and have advocated the application of solid penicillin to the infected ulcer. The present study on the

* Received for publication, July 7, 1947.

value of penicillin in the different varieties of hypopyon formation also attempts an assessment of subconjunctival injection as a method of application.

Clinical Data

1.—*Infected cornea ulcer with hypopyon*

1. *Infected corneal ulcer with hypopyon*.—Hardly any of the 39 cases of infected corneal ulcer with hypopyon conformed to the text-book picture of serpiginous ulcer. The ulcer itself was generally irregular in shape, the base and margin being infected to a variable degree; interstitial infiltration around the ulcer was present in 8 cases, whilst iritis was noted in 21 cases, though probably present in a subclinical form in all cases. In only one instance was the pneumococcus found. The clinical data of these cases are shown in Table I.

The following are the salient features:—

1. *Sex distribution*.—There were 30 men and nine women.
2. *Age distribution*.—This is shown in the following summary table.

Age					Number of Cases	
					M.	F.
0-9	—	—
10-19	2	1
20-29	1	—
30-39	3	—
40-49	2	2
50-59	10	—
60-69	9	5
70 and over	3	1

It will be noted that 28 out of the 39 patients were over 50 years of age.

3. *Trauma*.—In only 18 cases was there a clear history of antecedent trauma.

4. *Organisms*.—In 11 cases no bacteriological examination was carried out and in 9 more it was negative; the remaining 19 cases showed this distribution: staphylococcus albus 11, staphylococcus aureus 6, Morax-Axenfeld bacillus 1, pneumococcus 1.

5. *Degree of hypopyon formation*.—Using the arbitrary designations of minimal, $\frac{1}{4}$ of anterior chamber, $\frac{1}{2}$ and $\frac{1}{2}$ and total, the following distribution was observed:

Minimal	13 cases
$\frac{1}{4}$	22 "
$\frac{1}{3}$	2 "
$\frac{1}{2}$	1 case
Total	1 "

6. *Response to treatment.*—The following summary table shows that in 12 out of 39 cases there was a poor response to treatment or a relapse after initially satisfactory response. In the successfully treated cases recovery was generally rapid, judging by relatively short duration of in-patient treatment.

Treatment in days	Number of cases
up to 7 days	4
8 to 14 days	12
15 to 21 days	7
22 to 28 days	4
Poor response or relapse	12

7. *Response in relation to the patient.*—Three of the 12 patients with poor response or relapse, ultimately gave good functional results, 2 having vision of 6/6 and the third vision of 6/12. Two of these patients (Nos. 19 and 25) showed relapse; in the first patient after subconjunctival injections, in the second after oily drops. Ultimately a satisfactory end result was obtained by exclusive subconjunctival penicillin treatment. The third patient (No. 19) treated initially and unsuccessfully for 48 hours with subconjunctival injection, responded well to combined general sulphonamide and local penicillin therapy.

There are therefore 9 patients to consider. Of these the end results were fairly good in five in so far as useful vision was retained; in three more patients there was extensive opacification of the cornea, and 1 patient lost his eye altogether. Apart from the fact that these 9 patients contained a high proportion of women (4 out of 9) the age distribution and other clinical data were not dissimilar to those of the series as a whole. Three of these patients showed staph. aureus, 1 staph. albus, 1 no organisms and in 4 a culture was not taken. One patient had acne rosacea and one rheumatoid arthritis (Nos. 32 and 33 respectively). There was no history of trauma in three patients.

8. *Response in relation to the mode of treatment.*—As can be seen from Table I, subconjunctival injection was the main (or only) treatment in 21 cases. Other forms of local penicillin treatment were given in 12 more cases, whilst local penicillin treatment

combined with general sulphonamide therapy was employed in 4 cases. In 2 cases general sulphonamide therapy was used exclusively.

The following summary table shows the number of cases responding to treatment in relation to the mode of application.

Duration of in-patient treatment	Subconjunctival injection	Other local penicillin therapy			Combined penicillin and sulphonamide therapy	Exclusive oral sulphonamide therapy
		Concentrated ointment	Drops	Painting		
Up to 7 days ...	2	1	—	—	—	1
8 to 14 days ...	8	—	1	1	1	—
15 to 21 days ...	3	1	—	3	1	—
22 to 28 days ...	4	—	—	—	—	—
Poor response or relapse ...	4	1	1	3	2	1

Four failures out of 21 cases treated with subconjunctival injections compare favourably with 8 failures in 18 cases treated by other methods. The disproportion is, however, not quite so marked, for one of the failures with combined penicillin and sulphonamide treatment received subconjunctival injections. In all, 25 patients received subconjunctival injections (three also receiving sulphonamides simultaneously—Nos. 33, 34, 36, and one receiving it after unsuccessful application of drops—No. 25); five failed to respond satisfactorily, against 7 out of 16 treated initially by other methods.

That the advantage would indeed seem to lie with subconjunctival injections is suggested from a study of the cases showing poor response, and from the end results as regards vision for the series as a whole.

8. *Cases showing poor response.*—(a) *Poor response to subconjunctival injection* (Nos. 18—21). In two of these patients (Nos. 20 and 21) the end result was poor in spite of general sulphonamide therapy in both, and intramuscular penicillin therapy in one, after three days treatment exclusively with subconjunctival injections. In the two other cases the end result was good; in one case (No. 18) full vision was obtained by exclusive subconjunctival therapy after two relapses; in the last case (No. 19) a good result was obtained by the addition of general sulphonamide therapy.

(b) *Poor response to other forms of local penicillin therapy.*—(Cases Nos. 24, 25, 31, 32 and 33.) In one case, treated exclusively with concentrated penicillin ointment, the eye came to

excision (No. 24). In one case, treated exclusively with weak penicillin ointment, corneal scarring reduced vision to 6/24 (No. 31). In two further cases (Nos. 32 and 33) treated with weak penicillin ointment, oral sulphonamide had to be administered, combined in one case with one subconjunctival injection. In the one case in this series that did well (No. 25) with resultant vision of 6/12, a good response was obtained only after two subconjunctival injections.

(c) *Poor response to sulphonamide treatment.* (Nos. 36, 37 and 39). The end result as to vision in these three cases was H.M., 6/60 and 6/18. As these patients had received a variety of treatment in addition to the sulphonamide therapy (to which they did not respond) it is difficult to indicate which was the favourable factor in the one case with useful vision. It may, however, be of some significance that this patient (No. 36), is the only one of the three who had received subconjunctival injections.

10. *End-results as to vision.*—The following summary table shows the end obtained in the 18 cases treated exclusively by subconjunctival injections, compared with the 14 cases in which no such injections were used. In 7 cases (6 of which were cases with poor response) a variety of treatments had to be combined.

Vision at end of Treatment	Subconjunctival injections		Other methods
	Used exclusively	Together with other treatment	
6/9—6/5 ...	10	2	6
6/18—6/12 ...	1	2	2
6/60—6/24 ...	5 (a)	—	4 (c)
H.M.—P.L. ...	1	3	1
No. P.L. ...	—	—	1
Not recorded ...	1 (b)	—	—
	18	7	14

(a) 4 cases had pre-existing visual defect.

(b) Cornea clear.

(c) One had pre-existing visual defect.

It will be noted that in only 3 out of 18 cases treated exclusively by subconjunctival penicillin was corneal damage sufficiently

severe to reduce vision to less than 6/9. In contrast 7 out of the 14 cases treated by other methods gave an end result of vision less than 6/9.

2.—Hypopyon with herpetic or neuropathic corneal lesion

As can be seen from Table 11 there were 12 patients—9 men and 3 women—with hypopyon associated with a herpetic or neuropathic corneal lesion. There was an infected corneal ulcer in all but four cases, and the cornea was intact in only one case. All but one patient had some degree of iritis.

The hypopyon was minimal in 5 cases, occupied $\frac{1}{4}$ of the anterior chamber in 3, and $\frac{1}{3}$ of the anterior chamber in 4 more. In three patients herpes ophthalmicus was present; one patient had had an alcohol injection into the Gasserian ganglion for trigeminal neuralgia. Dendritic ulcer, or a history of it, was present in five patients. In the remaining three patients the cornea was insensitive. Treatment consisted of subconjunctival injections in 8 patients. Penicillin ointment 4-800 units per gram was used in 4 patients, in one of whom the ulcer was also painted with penicillin 2,500 units per c.c. Oral sulphonamide was used in 6 patients, "carbolicisation" with methyl salicylate in two, and concentrated penicillin ointment in 7 cases. Generally several modes of treatment were used in combination or in sequence.

In only two patients (Nos. 43 and 45) could the response to treatment be designated as good, though only in relation to the hypopyon and not as regards the end result for vision.

One ended in enucleation, and the corneae in the remaining cases all showed dense leucomata. Recovery was slow. The duration of in-patient treatment was generally prolonged as can be seen from the following summary table.

No. of inpatient days	No. of cases
Up to 7 days	—
8-14 days	1
15-21 days	1
22-28 days	3
29-35 days	3
36-42 days	3
43-49 days	1
50 days and over	3

3.—*Glaucomatous eyes with hypopyon*

In a series of 5 cases of old-standing glaucoma complicated by hypopyon (Table III), 3 eyes ended in evisceration or enucleation for rapidly developing panophthalmitis; 2 of these three eyes were treated intensively by subconjunctival injections of penicillin in addition to oral sulphonamide. One patient responded unexpectedly well to two subconjunctival injections of 50,000 units of penicillin followed by the instillation of penicillin ointment 8,000 units per gm. for 14 days. One further patient did not respond to penicillin "carbolicization" and the application of penicillin ointment 800 units per gram; there was a moderately good response to oral sulphonamide.

4.—*Hypopyon iritis*

Ten patients present 13 instances of hypopyon iritis, one patient having had a bilateral attack and recurrence in one eye, and another patient had recurrence of a unilateral attack (Table IV). Two of these ten patients showed infected corneal lesions. In them (Nos. 57 and 60), as in the remaining 8 patients, the essential lesion was, however, iritis, generally recurrent. In contrast to the sex distribution in patients with infected corneal ulcers, and with neuropathic keratitis (Tables I and II) there was no difference in the sex distribution in this group, men and women being equally represented. The hypopyon was minimal in 6 eyes, $\frac{1}{4}$ in 5, $\frac{1}{2}$ in one, and $\frac{3}{4}$ in one more.

As can be seen from Table IV response to treatment was good in all but one patient. However, it was only in relation to the hypopyon that response could be regarded as satisfactory. In relation to vision the results were not good in 8 out of the 11 eyes treated. This, however, must be ascribed to the pre-existing iritis rather than to the immediate attack of hypopyon formation, as is suggested by the fact that the 3 patients with a first attack (Nos. 61, 63 and 65)—all young people aged 26, 31 and 25 years respectively—recovered full vision.

The treatment adopted was atropine exclusively in 2 cases, oral sulphonamides in 2, subconjunctival injections in 4, combined local penicillin and oral sulphonamide therapy in 2, and these latter measures with additional measures in 3 more.

TABLE I.—INFECTED CORNEAL ULCER
Treated by Subconjunctival injection of Penicillin (50,000 units)

(a) Treated by Subconjunctival infection												
No.	Sex	Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Response	Days in hospital	No. of injections	Additional Penicillin Treatment in ointment form. Concentration U/gm. used	Treatment other than Penicillin	End result
1	M	66	Yes	Staph. albus...	—	+	Good	7	12	—	—	V-6/5
2	M	66	Yes	Morax-Axenfeld Bacillus.	Onyx	+	Good	28	14	—	—	Faint corneal nebula V-6/9
3	M	64	No	Staph. Albus.	—	Minimal	Good	16	2	—	—	V-6/9
4	F	47	Yes	Staph. Albus.	—	Minimal	Good	8	10	—	—	V-6/6
5	M	56	No	Staph. Aureus.	—	+	Good	14	7	25,000	—	Cornea practically clear. Died from operation for enlarged prostate
6	M	85	No	Staph. Aureus.	—	+	Good	10	10	25,000	—	Faint nebula central. V-6/12
7	M	54	No	Nil	Severe iritis	+	Good	24	2	25,000	—	Central corneal nebula V-6/9
8	M	68	Yes	Nil	—	1/3	Fair	25	20	50,000	—	—
9	M	34	No	Pneumococcus	—	Minimal	Good	12	16	50,000	—	—

TABLE 1.—continued.

No.	Sex	Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Response	Days in hospital	Treatment.			End result
									No. of injections	Additional Penicillin in ointment form. Concentration U/gm. used	Treatment other than Penicillin	
10	M	16	Yes	Not done	Interstitial infiltration	†	Good	12	16	50,000	—	V - 6/6
11	M	59	No	Staph. Albus.	Interstitial infiltration	Minimal	Good	12	10	100,000	—	Old nebula both V - 6/36
12	M	76	(Spastic entropion)	Staph. Albus.	Severe iritis	†	Good	14	15	100,000	—	Iris bound down Peripheral nebulae. V - 6/24
13	F	43	(Leucoma since childhood)	Not done	Moderate iritis	Total	Good	20	9	100,000	—	P.L. (as before)
14	M	59	No	Staph. Albus.	Interstitial infiltration	Minimal	Good	12	1	100,000	—	Old mustard gas keratitis. V - 6/36
15	M	40	Yes	Staph. Albus.	Interstitial corneal infiltration and mild iritis	Minimal	Good	7	4	100,000	—	Old nebula. Amblyopic V - 6/60
16	M	65	?	Nil	—	Minimal	Good	11	4	100,000	—	Barely perceptible scar V - 6/24

TABLE I.—continued.

No.	Sex	Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Response	Days in hospital	Treatment.			End result
									No. of injections	Additional Penicillin in ointment form. Concentration U/gm. used	Treatment other than Penicillin	
17	F	62	4 previous attacks; rheumatoid arthritis	Staph. Aureus.	Severe iritis	+	Fair	25	3	100,000	—	V 6/9
18	M	45	Yes admission	Staph. Albus.	—	+	Good but 2 relapses	7 16 9	18 6 12	50,000 50,000 50,000	—	Barely perceptible scar V 6/6
19	F	18	Yes	Nil	Severe iritis	+	Poor (to penicillin)	19	8	50,000	Oral sulphonamide additional to penicillin after 48 hrs	Barely perceptible scar V 6/6
20	M	32	Yes	Staph. Aureus.	Interstitial infiltration Severe iritis	+	Poor	77	19	100,000	Oral sulphonamide additional to penicillin after 3 days	Cornea perforated; leucoma adherens
21	M	63	Yes	Nil	—	+	Poor	57	12	100,000	Oral sulphonamide and intramuscular penicillin after 3 days	Cornea opaque

TABLE I—continued.
(b) Treatment by concentrated Penicillin Ointment.

No. Sex Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Treatment		Response	Days in Hospital	End result	
					Concentration used: U/gm.	Frequency of application			Central scar.	corneal V. = 6/60
22 M 58	Yes	Staph. albus.	Moderate iritis	†	100,000	3 hourly	Good	7	Central scar.	V. = 6/5
23 M 51	No	Not done	Mild iritis	Minimal	100,000	4 hourly	Good	18		
24 M 68	Yes	Staph. aureus	Interstitial infiltration with moderate iritis	†	50,000	Hourly for 2 days	Good at first. Relapse after two days. Rapidly developing panophthalmitis	12	Eye excised	
25 M 60	No	Nil	—	—	—	—	—	—	—	—
25 M 60	No.	Nil	—	†	10,000 (oil)	Hourly for 4 days	Poor	26	—	—
25a Second admission			—	—	(Relapse 10 days; cleared by two subconjunctival injections)		Good	—	V. = 6/12	
26 F 65	No	Not done	Mild iritis	†	2,500	†; †; 1 hourly, then 2 hourly	Good	11	Barely perceptible scar.	V. = 6/9

(c) Treatment by Penicillin Drops

TABLE I—continued.
 Treatment by daily "carbolicising" the Ulcer with Penicillin 2,500 units c.c. and subsequent application of dilute penicillin ointment (400-800 U/gm.).

No.	Sex	Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Treatment		Response	Days in hospital	End result
							Concentration of ointment used: U/gm.	Frequency of application of ointment			
27	M	52	No	Nil	—	Minimal	800	Hourly	Good	15	V. 6/9 Barely perceptible scar. V. 6/24
28	M	73	Yes	Nil	Moderate iritis	Minimal	800	Hourly	Good	10	Faint nebula. V. 6/9
29	M	52	Yes	Staph. albus	—	†	800	4 hourly	Good	19	Scar. V. 6/18
30	M	52	Yes	Nil	Mild iritis	Minimal	800	4 hourly	Good	19	V. 6/24
31	F	81	Yes	Not done	Severe iritis and interstitial infiltration	†	800	4 hourly	Poor; two relapses	27	Scar. V. 6/18
32	F	61	No (Acne rosacea)	Staph. aureus	Mild iritis	†	(a) 800 (b) Course of oral sulphamide	Hourly	Poor Good	19	Scar. V. 6/18
33	F	60	No (Rheumatoid arthritis)	Not done	Severe iritis	†	(a) 1,000 (b) One subconjunctival injection, and course of oral sulphamide	4 hourly by for 5 days followed by	Poor Poor	64	Central corneal scar. V. 6/36

TABLE 1—continued.
(e) Combined local Penicillin and general Sulphonamide Therapy.

No.	Sex	Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Mode of local penicillin therapy	Response	Days in hospital	End result
34	M	13	Yes	Staph. aureus	Severe iritis	1/3	Ung. 25,000 U/gm., 4 hourly, and subconjunctival injections 6 hourly	Good	15	V. = 6/9
35	M	69	No	Not done	Mild iritis	Minimal	12 instillations of gutt. 2,500 U/c.c. at 5 mins. intervals. Subsequently Ung. 800 U/gm. hourly	Good	12	V. = 6/9
36	M	58	Yes	Not done	—	†	(a) 27 subconjunctival 6 hourly. (b) T.A.B. subsequently	Poor	39	Moderate central opacity. V. = 6/18
37	M	22	Yes	Staph. aureus	Moderate iritis; interstitial infiltration	Minimal	(a) "Carbolizing" with penicillin 2,500 U/c.c. and Ung. 800 U/gm. hourly. (b) T.A.B. subsequently	Fair	34	V. = 6/60

(f) Exclusive General Sulphonamide Therapy.

No.	Sex	Age	History of trauma	Culture	Lesion other than infected ulcer	Hypopyon	Treatment	Response	Days in hospital	End result
38	M	33	Yes	Not done	Moderate iritis	†	Sulphamezathine	Good	7	V. = 6/9.
39	F	61	No	Not done	Iritis: corneal abscess on 12th day	†	(a) Sulphamezathine followed by Penicillin 800 U/gm. hourly (b) Ung. hourly	Poor	58	V. = 11 M.

TABLE II.
Neuropathic or herpetic keratitis with hypopyon.

No.	Sex	Age	History	Lesion	Hypopyon	Culture	Response	Days in hospital	Treatment (apart from atropine)	End result
40	M	64	Herpes ophthalmicus 2 years before	Infected ulcer, mild iritis	Minimal	—	Poor	35	(1) For 3 days daily "carbolyzation" with penicillin 2,500 U/c.c.; with penicillin 800 U/gm. hourly and methyl salicylate 4 hourly. Then (2) Oral sulphamezathine and cantharizing Ung Albucid.	Enucleation
41	M	82	Herpes ophthalmicus	Infected ulcer and mild iritis	1/3	Staph. aureus.	Poor	59	Oral sulphamezathine (for 4 days) together with ung. penicillin 800 U/gm. 2 hourly, and methyl salicylate cantharizing daily.	Desceemet's membrane removed on 14th day. Total corneal opacity. V.P.L.
42	F	84	Herpes ophthalmicus Spastic entropion	No corneal lesion. Severe iritis	+	—	Fair	34	Operation on entropion, and instillation of ung. penicillin 50,000 U/gm. 3 hourly. Also subconjunctival penicillin 50,000 units 3 in the first week and 2 each in the second and third weeks.	V 6/18 P.
43	M	34	Alcohol injection of Gasserian ganglion for tic douloureux	Infected ulcer and mild iritis	Minimal	Nil.	Good, but relapse 12 days later	26	(1) Three subconjunctival penicillin injections 50,000 units 6 hourly followed by ung. penicillin 100,000 units 4 hourly. (2) Seven more injections on relapse, followed by tarsorrhaphy.	Dense leucoma

TABLE II.—continued.

No.	Sex	Age	History	Lesion	Hypopyon	Culture	Response	Days in hospital	Treatment (apart from atropine)	End result
44	M	46	Recurrent dendritic ulcer. 4 or 5 attacks	Infected ulcer. Mild iritis	+	—	Poor	16	(1) Subconjunctival penicillin 50,000 units 4 hourly and ung. penicillin 100,000 U/gm. 4 hourly for 2 days. Then (2) Oral sulphamezathine 30 gms	Dense leucoma V = 2/60
45	M	76	4 months — persistent superficial punctate staining. Cornea anaesthetic	Infected ulcer. Mild iritis	+	—	Good	9	12 subconjunctival injections of penicillin 50,000 units 6 hourly.	V = P.L.
46	M	49	5 years — intermittent dendritic corneal ulceration. 3 weeks — recurrence	Infected ulcer	Minimal	Nil.	Poor	51	In succession: (1) 12 subconjunctival injections and penicillin 50,000 U/c.c. 6 hourly, and ung. penicillin 100,000 U/gm. 4 hourly. (2) Oral sulphamezathine 30 gms. (3) Milk injections. (4) Short wave therapy.	Dense leucoma
47	F	61	10 months previously a dendritic ulcer. Started as iritis and developed corneal stain on 24th day of treatment	Interstitial infiltration. Moderate iritis	Minimal	—	Poor	35	(1) Ung. penicillin 400 U/gm. 4 hourly for 6 days. Then (2) Sulphamezathine 30 gms.	Spontaneous improvement after cessation of all treatment

TABLE II.—continued.

No.	Sex	Age	History	Lesion	Hypopyon	Culture	Response	Days in hospital	Treatment (apart from atropine)	End result
48	M	19	Gradual onset over 3 weeks. Poor sensation	Interstitial infiltration resembling disciform keratitis. Moderate iritis	Minimal	—	Poor	25	(1) 20 subconjunctival injections of penicillin and ung. penicillin 100,000 U/gm. 4 hourly. (2) Tarsorrhaphy.	Relapse V. 6/36. Under treatment
49	M	57	One week gradual onset of pain in eye. Cornea insensitive	Infected corneal ulcer and severe iritis	1/3	Nil.	Poor	46	20 subconjunctival injections of penicillin 6 hourly, and ung. penicillin 100,000 U/gm. 4 hourly.	Total corneal opacity. Iris bound down. V. P.L.
50	M	60	Similar attack 10 years ago. Cornea insensitive. Sore 3 weeks	Infected ulcer and severe iritis	1/3	—	Poor	54	Ung. penicillin 800 U/gm. hourly and drops 2,500 U/c.c. at 15 minutes—1 hour for 8 days. Subsequently oral sulphamezathine.	Dense leucoma
51	F	60	Tarsorrhaphy in the past. Broken down corneal scar; 3 days later hypopyon ulcer. Cornea insensitive	Interstitial infiltration and moderate iritis	1/3	Nil.	Poor	24	Ten subconjunctival injections of penicillin, and ung. penicillin 100,000 U/gm. 4 hourly. Subsequently repeated. A.C. washed out and milk injections.	Dense leucoma

TABLE III.
Glaucomatous eyes associated with hypopyon.

No.	Sex	Age	History	Culture	Lesion	Hypopyon	Response	Days in hospital	Treatment	End result
51	F	85	For 5 years a blind glaucomatous eye	—	Large corneal abscess	+	Poor	18	(1) Penicillin drops 2,500 U/c.c. $\frac{1}{4}$ hourly for 6 hours followed by ung. penicillin 800 U/gm. $\frac{1}{4}$ hourly for 5 days. Albucid drops subsequently for 4 days	Enucleation on 9th day
52	M	62	Old iritis, secondary glaucoma	—	Iris bombé	+	Poor	32	Twelve subconjunctival injections of penicillin followed by 3 intravitreal injections 5,000 units penicillin and oral sulphamezathine	Evisceration
53	M	41	Bilateral buphthalmos c. iridectomy at age of 10. Eye hit with twig 5 days ago	Staph. aureus at evisceration	Infected corneal ulcer	+	Poor	48	(1) 44 subconjunctival injections together with ung. penicillin 100,000 units 2 hourly. (2) On 4th and 6th days intravitreal injection of penicillin 5,000 units with sulphapyridine from 4th day onwards. Paracentesis on 6th day.	Evisceration. Hypopyon had disappeared after 24 hours but panophthalmitis rapidly developed
54	F	64	A blind eye due to chronic glaucoma	Pneumo. cocci	Infected ulcer and moderate iritis	+	Good.	37	Two subconjunctival injections penicillin 50,000 units. Ung. penicillin 8,000 U/gm. hourly	Corneal nebula
55	F	74	Blind eye from long standing chronic glaucoma.	Nil.	Infected ulcer and mild iritis	+	Good	19	(1) "Carbolization" with penicillin 2,500 U/c.c. and ung. penicillin 800 U/gm. hourly for 3 days. Then (2) oral sulphamezathine	Corneal nebula

TABLE IV—Hypopyon Iritis

No.	Sex	Age	History	Lesion other than iritis	Hypopyon	Response	Days in hospital	Treatment (apart from atropine)	End result
57	F	40	Recurrent bilateral iritis. Congenital spastic paraplegia	Interstitial infiltration right cornea	1/3	Poor	40	In succession:—(1) Subconjunctival penicillin 50,000 units 6 hourly, and Urog. penicillin 25,000 U/gm. 4 hourly for two days. (2) Oral sulphamezathine. (3) Short wave therapy.	R.V. = P.L. L.V. = 6/60
58	F	82	Influenza 14 days before admission. Old rheumatoid arthritis	—	+	Good	15	(1) Two subconjunctival injections of penicillin 50,000 units; followed by oily penicillin 10,000 U/c.c. 2 hourly	Cornea clear. Atrophic iris. V. = H.M. Lens opaque
59	F	27	Recurrent bilateral iritis	—	Right minimal. Left +	Good	21	Subconjunctival penicillin 50,000 units 4 hourly for 4 days	R.V. = 5/60 L.V. = C.F. at 1m.
59a	Second admission		ditto	—	Right minimal	Good	6	As before	—
60	F	63	Diabetic iritis, rubeosis and secondary glaucoma	Infected corneal ulcer	+	Good	22	(1) Subconjunctival penicillin 50,000 units 6 hourly, and Urog. penicillin 100,000 U/gm. 4 hourly for 3 days, followed by (2) Oral sulphamezathine and short wave therapy	—
60a			ditto	ditto	+	Good	26	(1) Subconjunctival penicillin 50,000 units 6 hourly, and Urog. penicillin 100,000 units 4 hourly for 3 days followed by (2) Short wave therapy	Dense corneal scar. Occluded pupil

TABLE IV—Hypopyon Iritis—(continued).

No.	Sex	Age	History	Lesion other than iritis	Hypopyon	Response	Days in hospital	Treatment (apart from atropine)	End result
61	M	26	Acute iritis	—	Minimal	Good	18	Ung. penicillin 8,000 U/gm. 4 hourly and oral sulphamezathine simultaneously	Eye normal. V. 6/6
62	M	8	Endophthalmitis following an infectious illness a year ago. Blow on eye 4 days ago	Endophthalmitis	Minimal	Good	43	(1) Two subconjunctival injections of penicillin 50,000 units; also ung. penicillin 25,000 U/gm. 2 hourly, with oral sulphamezathine simultaneously	Shrunken eye
63	F	31	Acute iritis	—	Minimal	Good	8	Oral sulphamezathine	Eye normal. V. 6/6
64	M	61	Recurrent iritis	—	Minimal	Good	10	Sulphamezathine 30 gms., mydrinate and short wave therapy	V. = 6/18
65	M	25	Acute iritis	—	†	Good	19	Heat	Eye normal. V. 6/6
66	M	57	Left iritis 5 years ago. Recurrence now with hypopyon	—	†	Good	0	Mydrinate daily	Hypopyon disappeared within 3 days. V. 6/24

Discussion

1. *Causes of hypopyon formation.*—Hypopyon as a complication of infected corneal ulcer, and hypopyon associated with iritis are clearly recognised as distinct clinical entities. The fundamental difference is, of course, that in the first group the cornea is the seat of the primary lesion, and in the second the iris lesion is the responsible factor. This distinction is important and generally valid, but the presence of a corneal lesion in two of the cases of hypopyon iritis (Nos. 57 and 60) shows how difficult it may be in individual cases to assess the type of hypopyon with which one is dealing—a difficulty all the more real as severe iritis is not infrequent in hypopyon corneal ulcers. The diagnostic difficulties are complicated still further by the occurrence of hypopyon in the group of cases shown in Table II. Both iritis and corneal ulcer were present in most of these cases, which, however, constitute a distinct entity of their own in that the primary lesion appeared to be neither of these affections, but loss of corneal sensation of a trophic or possibly virus origin. This group does not appear to be well recognised, but the therapeutic response no less than the disturbed corneal sensation leave no doubt that it must be regarded as distinct from the others. In a final group, glaucomatous degeneration of the eye appeared to have been the primary disturbance, with the hypopyon formation as the result of either an iris reaction or a corneal lesion.

As distinct from these four types, there were cases of hypopyon formation following an operation, or on infection carried into the eye by trauma. These cases have not been considered in the present study because their fulminating course and response to treatment would suggest that they are true infections as distinct from aseptic inflammatory reactions and require detailed consideration on their own.

2. *Response to treatment.*—It is clear from the data in Tables I-IV that generally speaking infected corneal ulcers (as tabulated in Table I) respond well to local penicillin therapy, and that hypopyon formation secondary to herpetic or neuropathic corneal lesions gives no response. It would also seem that whether a response is obtained in hypopyon formation in glaucomatous eyes depends upon the condition of the eye, and possibly also whether the hypopyon is septic in origin. In contrast to the efficacy of penicillin in infected corneal ulcers, and its uselessness in the herpetic and neuropathic hypopyon formation, is the lack of need of penicillin in hypopyon iritis. From the data shown in Table IV it is seen that hypopyon iritis responds well to treatment by atropine, and nothing else is required.

Reduced to general terms one may perhaps say that an infected

corneal ulcer is sterilised by the action of penicillin; this explains the value of penicillin in these cases—the hypopyon disappears when the primary infection is brought under control. In contrast, hypopyon formation secondary to virus or neuropathic corneal lesion does not respond to penicillin, presumably because the virus is penicillin-resistant, and that in a neuropathic lesion there may be no infecting agent at all. Finally in hypopyonitis control of the inflammatory iris reaction by atropine brings about absorption of the hypopyon; penicillin is unnecessary as there is no bacterial exciting factor. As in infective lesions generally, so in hypopyon reactions, penicillin can give results only when the exciting cause is infective in character and susceptible to penicillin.

3. *Mode of use of penicillin: theoretical considerations.*—It has been shown elsewhere (Sorsby and Ungar, 1946) that subconjunctival injection of 25,000 units of penicillin gives higher and more sustained aqueous levels than the administration of 50,000 units intravenously, or 40,000 units in beeswax intramuscularly, or of the insertion into the conjunctival sac of ointment containing 100,000 units per gm. The simpler procedures of the instillation of drops, and of ointments containing a low concentration of penicillin are inapplicable if an adequate intra-ocular level of concentration is to be reached (Struble and Bellows, 1944). Nothing is known of the concentration in the aqueous on the application of penicillin in solid form to the surface of an infected corneal ulcer as advocated by Juler and Young.

On theoretical grounds it would therefore seem that subconjunctival injection is the method of choice for obtaining and maintaining high intra-ocular levels of concentration of penicillin. It is superior to both systemic administration and to other methods of local application. The detailed studies of Andrews (1947) and of Sorsby and Ungar (1947) have confirmed the high intra-ocular levels of concentration reached by this mode of administration of penicillin. It would furthermore appear that the addition of adrenalin to the penicillin materially increases both the levels reached and the levels maintained, as can be seen from the following summary table (Sorsby and Ungar, 1947).

Level of penicillin in the aqueous and the cornea of the rabbit after subconjunctival injection of 50,000 units pure penicillin with and without adrenalin

Hours	$\frac{1}{4}$	$\frac{1}{2}$	1	2	3	4	6
Aqueous. Without adrenalin	24	19	17	9	3	0.75	0.56
Aqueous. With adrenalin ...	>32	>17	>32	>32	>20	20	1.25
Cornea; Without adrenalin	950	97	76	25	18.75	4.87	1.5
Cornea. With adrenalin ...	>860	>200	>1,440	925	450	65	2.5

The clinical use of massive doses of penicillin subconjunctivally only became possible with the advent of purified penicillin. With such penicillin, injections of 50,000 units are well tolerated, even when repeated frequently. (Sorsby and Ungar, 1946.)

4. *Management of hypopyon corneal ulcer susceptible to penicillin.*—Theoretical expectations are borne out by the results recorded for the cases summarized in Table I. Subconjunctival injection of penicillin proved superior to several other modes of applying penicillin. The relative value of subconjunctival penicillin and oral sulphonamide cannot be assessed, as no substantial studies on the effect of the sulphonamides on hypopyon keratitis appear to be available.

As this work was progressing a standard method of treatment began to emerge. At the moment the routine used consists of the following steps:

(1) After a smear is taken for culture purposes the eye is irrigated with half normal saline at room temperature to remove any organic matter.

(2) The conjunctiva is anaesthetised by the instillation of two drops of 4 per cent. or 5 per cent. cocaine hydrochloride solution, repeated if necessary.

(3) Into an ampoule containing 100,000 units of white crystalline penicillin 0.5 c.c. of 2 per cent. novocaine and 0.5 c.c. of adrenalin 1:1,000 is injected. 0.5 c.c. is now withdrawn and injected subconjunctivally.

(4) Drops of atropine are instilled into the conjunctival sac and the eye is bandaged.

(5) Injections are repeated at 6-hourly intervals for three days, so that a total of 12 injections are given in all cases. Where response appears to be halting another four injections are given over an additional day.

(6) When injections are suspended penicillin ointment in a concentration of 100,000 units per gm. is instilled at four-hourly intervals day and night. The ointment should be made up with a specially prepared base of petroleum jelly and liquid paraffin 90 and 10 parts of each respectively. The penicillin is incorporated into this base without dissolving it in water. The ointment is continued for 48 hours after apparent clinical cure.

Whilst normally the solvent for the penicillin injected subconjunctivally is an equal quantity of 1:1,000 adrenalin and of 2 per cent. novocaine, it is possible to use either solvent by itself. Novocaine helps to overcome the discomfort some patients experience on repeated subconjunctival injections; adrenalin is

well tolerated by most patients. Sterile water can be used, but saline should not be employed as it produces a hypertonic solution which is distinctly uncomfortable on injection. It is feasible to replace either of these solvents by mydracaine where the iritis is particularly intense. Adrenalin solution, because of its low pH, tends to destroy penicillin in vitro (Cameron, 1945). Clinically this deleterious effect does not seem to be substantial; in the tissues subconjunctivally the adrenalin is apparently rapidly buffered; in solution in vitro the amount of penicillin destroyed would appear to be small.

Where no substantial response is obtained with subconjunctival injections at the end of 48 hours, further treatment on these lines is useless.

Subconjunctival injections of penicillin as a method of treating hypopyon formation secondary to infected corneal ulcer give gratifying results. The management of the occasional resistant case is still a problem of some difficulty. A full exploration of the possibilities of oral sulphonamide would seem to be desirable.

Summary

1. Results of treatment, mainly with penicillin, are recorded for 66 patients with hypopyon.

2. This series included 39 cases of infected corneal ulcer; 18 of these were treated by subconjunctival injections of penicillin in doses of 50,000 units with or without the application of penicillin ointment in concentrations of 25,000 to 100,000 units per gm. Other methods of local penicillin therapy were employed in 12 patients. Oral sulphonamide treatment was used in two cases and local penicillin therapy combined with general sulphonamide therapy in four more. In three patients general sulphonamide therapy was used when subconjunctival injections of penicillin proved inadequate.

Twelve patients with hypopyon formation associated with herpetic or neuropathic corneal lesions were treated by various applications of penicillin locally, with or without general sulphonamide therapy.

There were also five cases of old-standing glaucoma showing hypopyon as a complication.

A final group of 10 patients presented 13 instances of hypopyon iritis.

3. Infected corneal ulcers responded well to treatment. Hypopyon seen in herpetic or neuropathic keratitis gave no response. Hypopyon iritis appeared to require no treatment other than atropine.

4. A detailed analysis of the cases of infected corneal ulcers with hypopyon treated by different methods of penicillin therapy shows subconjunctival injections to be the method of choice. It was successful in 18 out of 21 cases so treated.

5. The mode of treatment is described. It consists essentially of 12 to 16 subconjunctival injections each of 50,000 units of penicillin (dissolved in 0.25 c.c. of 2 per cent. novocaine and 0.25 c.c. of 1:1,000 adrenalin) at intervals of six hours, followed by the 4-hourly instillation of penicillin ointment in a concentration of 100,000 units per gm.

Our thanks are due to Mr. Savin, Mr. Rycroft, Mr. Tyrrell, Miss Dollar and Mr. Cameron of the Royal Eye Hospital for help with clinical material. We are indebted to successive House Surgeons for their help, and to Sisters Bradley and Hasfield of the Royal Eye Hospital, and Sisters Hollands and Thorogood of the Lambeth (L.C.C.) Hospital Ophthalmic Unit for their loyal and painstaking collaboration.

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THE LAMINA CRIBROSA AND ITS NATURE*

BY

Professor MARIAN WILCZEK

CRACOW

THE present knowledge of this subject is based mostly on the work of E. Fuchs (1916) as there have been no newer researches in this field. The cribriform lamina is generally taken to be a semi-independent structure. The dimensions of its anterior and posterior planes, its thickness and backward curve and other dimensions have been already measured. In cases of glaucoma the possibility of the cribriform lamina being pushed backward by the raised intra-ocular pressure has been generally accepted.

Birnbacher and Czermak (1885—1886) have likened the cribriform lamina to a membrane of India-rubber which can bulge under

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intra-ocular pressure. According to the generally accepted opinions the cribriform lamina is a part of the sclera. Lauber in his textbook, published in 1936, writes: "The internal layers of the sclera form in the scleral canal a network of lamina cribrosa sclerae; strictly speaking it is only the thinned part of the sclera." E. Fuchs states: "Part of the fibres of most internal layers of the sclera is stretched over the foramen sclerae . . . this part when cut looks like a sieve, and therefore it is called lamina cribrosa."

The opinions of other authors are almost similar. These views are not necessarily strictly accurate. For instance, in a case of deep physiological depression of the optic disc one sees through the ophthalmoscope the cribriform lamina lying 1—1.5 mm. below the level of the retina. Now it is known that the sclera in this region is 1—1.5 mm. thick. Round the optic nerve it becomes thinned to one-half or one-third by the inter-meningeal recess (*recessus intravaginalis*). So it can hardly be maintained that the cribriform lamina is really a thinned part of the sclera. In this case at least posterior trabeculae of the cribriform lamina must be derived from the pia mater of the optic nerve and not from the sclera.

Rather complicated relations in the arrangement of neuroglia in the optic disc, in the first segment of the optic nerve and in the insertions of meningeal sheaths of the nerve to the sclera can be understood only in view of certain facts derived from the development of the eye. The publications of Seefelder (1910), Kleczkowski (1913) and v. Szily (1921—1922, 1930) have the greatest bearing on this subject. All changes leading to the conversion of the optic vesicle into the optic cup aim at creating the best connections of nerve-fibres of the retina with the brain by forming the embryonic disc of the optic nerve (*papilla primitiva s. epithelialis*). Nerve-fibres of the retina grow into this embryonic disc, and so there develops the definite optic disc, collecting into a bundle all nerve-fibres of the retina. Cells of the stalk of the cup convert into neuroglial cells, and so neuroglial stroma of the optic disc and nerve are developed. Bundles of nerve-fibres are separated one from another by neuroglial tissue which forms the first septa of the optic disc and nerve. Only later—as Kleczkowski has shown—connective cells grow into these neuroglial septa, and supply the nerve with vessels. Connective tissue divides the nerve into smaller and smaller bundles forming first-, second- and third-grade septa; but still smaller septa are formed by neuroglia.

Bearing this in mind, one may understand the relations between neuroglial and connective tissue. Neuroglia completes connective septa and carries on the partition into still smaller bundles, forms a sheath between the walls of the scleral canal and the disc (*inter-mediäres Gewebe*), invests anteriorly central connective tissue

which envelops the vessels (Zentr. Bindegewebsmeniskus Kuhnt), penetrates into the disc along the vessels (Schaltgewebe), forms the so called neuroglial cribriform plate (lamina gliosa), and envelops each connective septum in the cribriform lamina and in the nerve. All these details in distribution of neuroglia are fairly clear, if one remembers that the stalk into which nerve-fibres have eventually grown, was of neuroglial origin, and that connective tissue has grown into primitive neuroglial septa, rather strengthening them than eliminating them completely.

All connective coats of the eye, such as the choroid, the sclera, meningeal sheaths of the optic nerve, septa of the optic nerve and the cribriform lamina are formed from the same mesenchymal mass, which envelops the embryonic eye when the retina, the optic disc and nerve have already been formed. That is why there is no sharp division between pia or dura mater and the sclera, but meningeal sheaths pass into and coalesce with the sclera.

In order to be thoroughly acquainted with the structure of the cribriform lamina, its relation to the sclera and septa of the optic nerve, with its function and importance, I have carried out a series of my own investigations. I have studied histological longitudinal and transverse sections of the cribriform lamina, not only of adults, but also of the newly-born and embryos. I have also studied the structure of the cribriform lamina of the following animals: cat, pig, horse, and ape. The examination of the complete series of transverse sections seems to me most instructive.

When examining the preparations from different periods of the development of the eye, I could demonstrate that the cribriform lamina develops comparatively late, and its complete development is attained as late as the end of foetal life or even at the beginning of extra-uterine life. Trabeculae of connective tissue grow into the nerve and the disc from all directions and from all available sources of connective tissue, namely from the pia mater, from the sclera in the scleral canal and—as can be demonstrated in the series of transverse sections—also from connective tissue, which accompanies the central vessels. These connective trabeculae form the septal network of the nerve. By their ingrowth the nerve is supplied with vessels and its structure strengthened, delicate nervous tissue connected with the pia mater and the optic nerve inside the scleral canal fixed to the sclera.

In the embryonic eyes, where there is as yet no cribriform lamina and no connective septa of the nerve, it can sometimes be observed how the optic nerve slips out of the scleral canal (Fig. 1). Trabeculae which grow into the nerve from all sources of connective tissue in this region must be stronger, the more abundant the source of connective tissue. In that way one should explain the

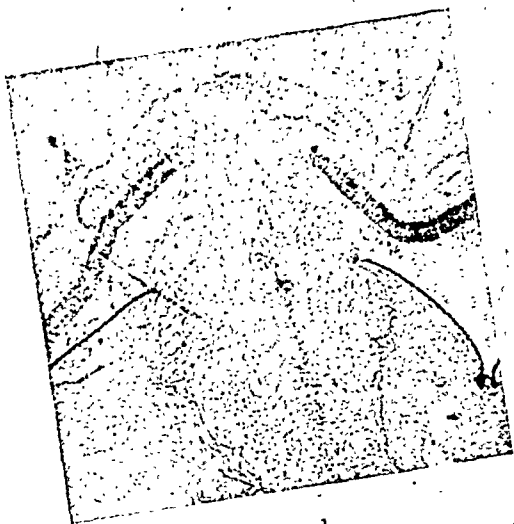


FIG. 1.

The optic nerve in a human embryo, 6 months old, which has not been attached to the scleral canal by the cribriform lamina, and has slipped out of the canal.

fact that trabeculae in the cribriform lamina are much thicker than in the nerve, for septa of the nerve come from the comparatively thin-pia mater, whereas trabeculae of the cribriform lamina come from abundant connective tissue of the sclera. Sometimes one can notice delicate trabeculae penetrating into the optic nerve from the choroidealis. These trabeculae are rather scanty and thin, which can be explained by the fact that the choroid is rather a poor source of connective tissue. I consider fixation of the optic nerve in the scleral canal, i.e., in the place where the optic nerve is exposed to the great danger of being pulled, as one of the most important functions of the cribriform lamina.

During my studies of the cribriform lamina I have made the first plastic model of this region of the eye which enables one to visualize the relations in three dimensions. This model has been constructed in the following way: from a complete series of histological sections of the optic nerve of a normal human eye—sections being 20 microns thick—I chose the sections comprising the whole scleral canal from the anterior plane of the sclera to the first segment of the optic nerve surrounded by the intermeningeal space. I took microphotographs of each section and magnified them 50 times on paper, under which I inserted a layer of mixture of paraffin and beeswax, 1 mm. thick. Thereby I obtained a series of plates, each corresponding to a microscopic section.

magnified 50 times. From these plates, using Graefe's knife and Gullstrand's binocular, I excised all the outlines of nerve-bundles, leaving only septal network of connective tissue. In this way I obtained the isolated connective scaffolding of the nerve surrounded by the sclera or pia mater. By sticking all the plates together I obtained a model which enabled me to study carefully all the details. While looking at the transverse sections of my series and putting the model together I became convinced that there is no substantial difference between the cribriform lamina and the septal system of connective tissue of the optic nerve. They actually form one common system whose parts can be differentiated only by their point of origin. In the scleral canal, trabeculae are thicker because they come from the sclera; in the nerve they are thinner because they come from pia mater.

The cribriform lamina is then not an independent structure but the foremost and strongest part of the septal system of connective tissue of the nerve.

Fig. 2 shows a section, 89, of my series. One can see in the upper part the septal network coming from the wall of the scleral canal; it is the cribriform lamina. In the lower part of this preparation, trabeculae must come from pia mater as the intermeningeal space can be clearly seen. These trabeculae belong to the septal system of the nerve. There is no difference between these two systems. They both form a uniform network whose trabeculae are



FIG. 2.

In the upper part of the preparation notice a trabecular network of the cribriform lamina coming from the sclera, in the lower part the septal network of the optic nerve coming from pia mater.

a little thicker in the part which has to be included in the cribriform lamina. On the whole one can compare this septal system to the canals of connective tissue in whose walls there are numerous gaps. The canals may join the neighbouring one or divide.

Fig. 3 represents the front view of the model. In the foremost part of the scleral canal there is an apparent shallow depression, at the bottom of which one may see the front of a septal system, i.e., the cribriform lamina. This depression, demonstrated for the first



FIG. 3.

The plastic model viewed from the front. Notice the depression called by the author "the nest of the optic disc."

time on my model, should be called the nest of the optic disc (*nidus seu nidulus papillae nervi optici*), because in this very depression lies the optic disc. The bottom of the nest is uneven, slightly knob-like. Trabeculae of the cribriform lamina lie there but not on the same level, some being a little higher than the others. Moreover some trabeculae come from the walls of the nest, sometimes even from the anterior surface of the sclera. From the bottom of the nest there arises towards the front a fairly thick bundle of connective tissue investing the central vessels. In the disc short trabeculae come from this bundle and join the tissues of the disc. As the diameter of the nerve increases with its further course, the lateral walls of the nest are inclined and divergent. The shape of the nest may change according to the shape of the optic disc. All the dimensions of the nest and the inclination of its walls can vary to a certain extent.

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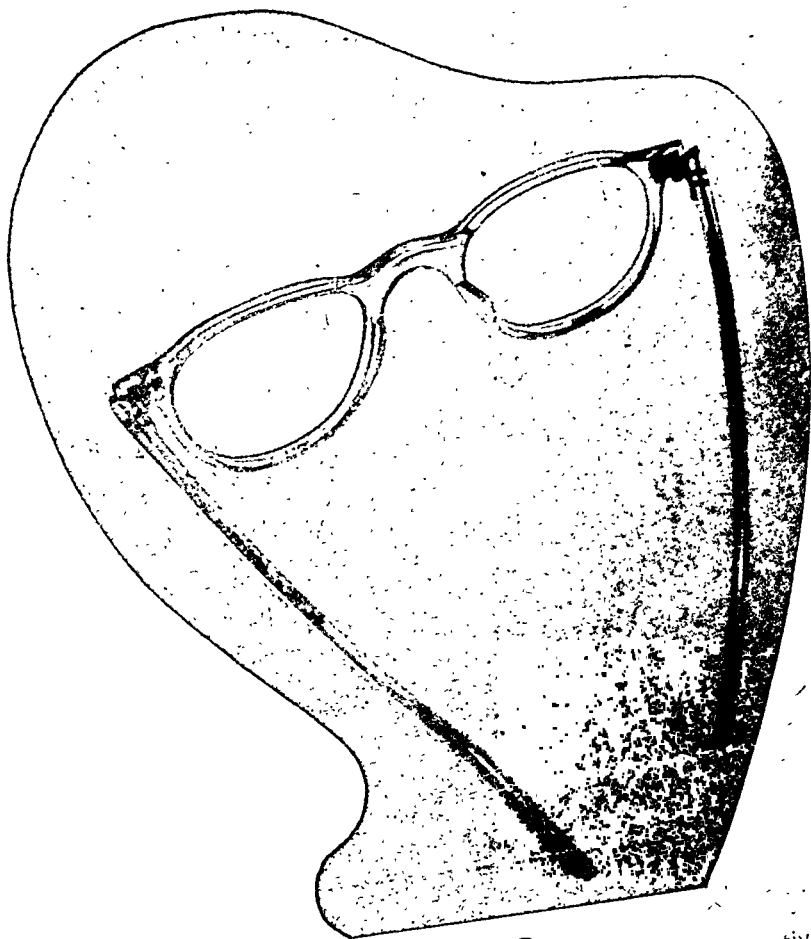
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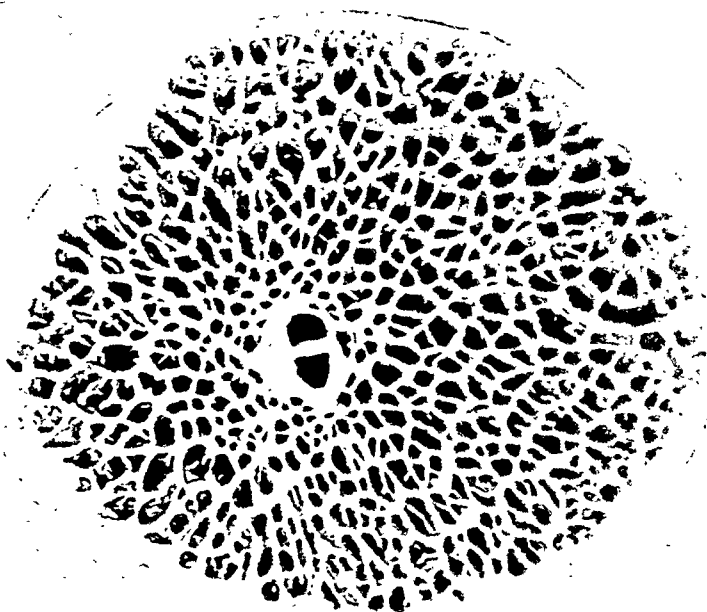


FIG. 4✓

The model viewed from behind. Septal system of the optic nerve.

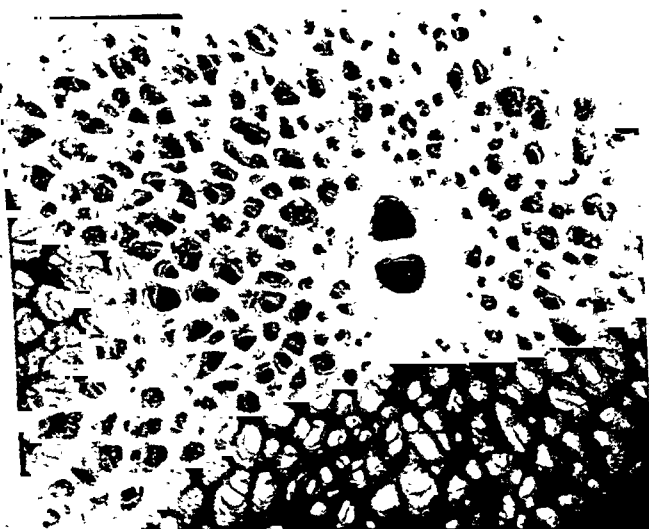


FIG. 5.

The model transilluminated from behind.

In my opinion introduction of the concept of the nest of the optic disc is advisable as much from the anatomical as the clinical point of view. This concept will permit more synthetic representation of the shape of the foremost part of the scleral canal.

Fig. 4 represents the septal system of the optic nerve viewed from behind in my model. Fig 5 part of the model illuminated from behind. One can see the interior of the canals in which there were bundles of nerve-fibres. Figs. 6 and 7 represent the model cut in the sagittal plane. One can see for the first time the outlines of septa at the same time in transverse and longitudinal sections.

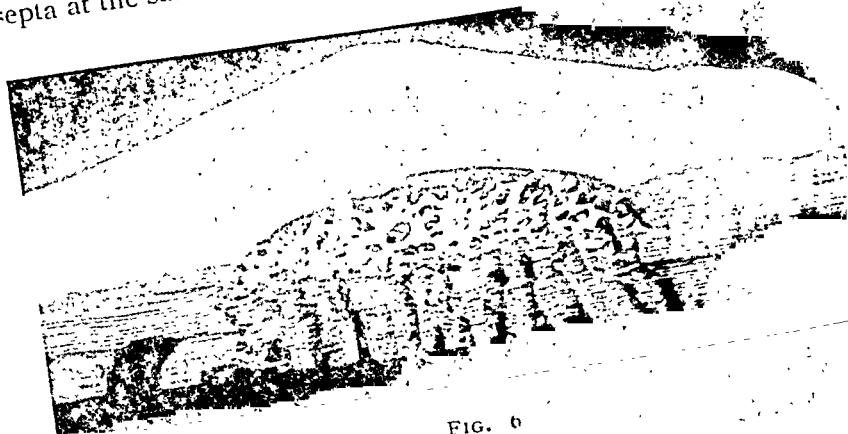


FIG. 6

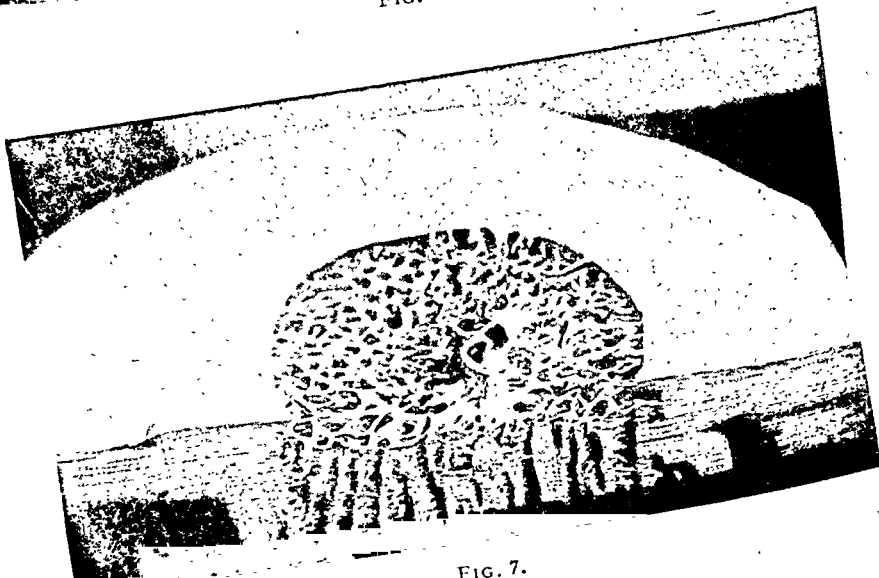


FIG. 7.

The relations between nervous and connective tissue are changing at various levels of the optic disc and nerve. In the disc there is almost complete lack of connective tissue. Beyond the cribriform lamina septa become thinner whereas bundles of nerve-fibres, developing myelin sheaths, become thicker. In order to determine strictly these relations I took the following measurements: from the microphotographs of the sections, magnified 50 times, I cut out with Graefe's knife the outlines of nerve bundles—as I did when making the model—leaving only the network of connective tissue. Then I cut out the neighbouring scleral tissue, the meninges and the outlines of the vessels. So, using the balance, I could strictly determine the relation between connective and nervous tissue in a given section. Then I calculated the area of the section, and knowing the percentage relation of the tissues and the magnification, I could easily calculate the real area occupied by each tissue in a given section. As I knew the number of holes, occupied by nerve-bundles in each section, I could calculate the average size of a nerve-bundle and the thickness of a septum of connective tissue. For my calculations I chose the sections at different levels of the disc, the cribriform lamina and the optic nerve. These are the sections of my series labelled 61, 65, 75, 79 and 95. Section 61 passes through the first part of the scleral canal in which there are no septa of connective tissue yet and nervous tissue still retains the structure of the disc. In the periphery of the section 65 there are already some short trabeculae of septa. Section 75 passes through the anterior part of the cribriform lamina, section 79 through the strongest part of the cribriform lamina, section 95 through the first part of the optic nerve, surrounded by the intermeningeal space. The results of these calculations are shewn on the Table I:—

Section	Area of section	Area of nervous tissue	Area of connective tissue	Nervous tissue in per cent.	Conn. tissue in per cent.	Amount of nerve bundles
61	1'35mm. ²	1'28mm. ²	—	per cent. ca.95	—	—
65	1'60 "	1'28 "	—	ca.80	ca.20	—
75	2'44 "	1'09 "	1'35mm. ²	44'7	55'3	350
79	2'50 "	0'9 "	1'60 "	36	64	394
95	3'10 "	1'78 "	1'32 "	57'4	42'6	398

It follows from this table that the area of section of the optic disc is 2.3 times smaller than the area of section of the first part of the optic nerve situated only 0.7 mm. below the former (sections 61 and 95). In the disc, 95—80 per cent. of the section is occupied by nervous tissue, compared with 36 per cent. in the most strongly developed part of the cribriform lamina where the remaining area is occupied by connective tissue. The figures of Table I can represent a real basis for any inferences concerning endurance of this area to endocular pressure, to stretching and to mechanical distortions, which occur in inflammatory processes, oedema, etc. From column 2 one can see that nervous tissue in the disc occupies greater area than in the cribriform lamina. As the amount of nerve-fibres is the same in both cases it follows that nerve-fibres in the disc lie more loosely, embedded in soft neuroglia. In the anterior layer of the cribriform lamina (section 75) the area of nervous tissue is a little greater than in the strongest part of the cribriform lamina (section 79) where nerve-fibres lie most tightly. Here, and not in the anterior opening of the scleral canal nor in the opening of lamina elastica choroidealis—as could be inferred from the longitudinal histological sections—there is the narrowest passage for nerve-fibres.

That is in the cribriform lamina that nerve-fibres can be most easily damaged by inflammatory processes, oedema, hyperaemia of trabeculae of connective tissue, etc.

Fig. 8 represents these relations in their true proportions, according to the calculations in Table I. The whole mass of nervous tissue lies in the centre of the drawing; it has the shape of an hour-glass. On each side there is a mass of connective tissue which varies in strength in different sections. In column 3 of Table I, section 75 contains nearly the same amount of connective tissue as section 95 which corresponds to the first segment of the optic nerve. It can be inferred that septa in the first segment of the

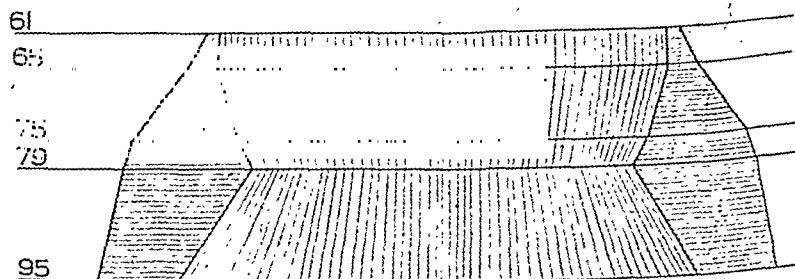


FIG. 8.

optic nerve are thinner than these in the first segment of the cribriform lamina not as a result of the smaller amount of connective tissue in the first segment of the nerve, but because these septa have been stretched by the bundles of nerve-fibres becoming thicker under the influence of myelinisation. The amount of connective tissue in the strongest part of the cribriform lamina is only 18.5 per cent. greater than in the anterior part of the cribriform lamina.

If one knows the area of connective and nervous tissue in the section and the amount of nerve-bundles one can calculate as well the average area and the diameter of a single nerve-bundle as the thickness of, surrounding connective tissue. I calculated it for sections 79 and 95 (strongest part of the cribriform lamina and first segment of the nerve). The results are given on Table II:—

Section	Area of one nerve-bundle	Diameter of one nerve-bundle	Area of one nerve-bundle together with connective tissue surrounding it	Difference of diameter i.e., thickness of the sheath of connective tissue between nerve-bundles
79	0.00225mm ²	0.0534mm	0.00625mm. ²	0.0346 mm.
95	0.00445 „	0.075 „	0.00775 „	0.024 „

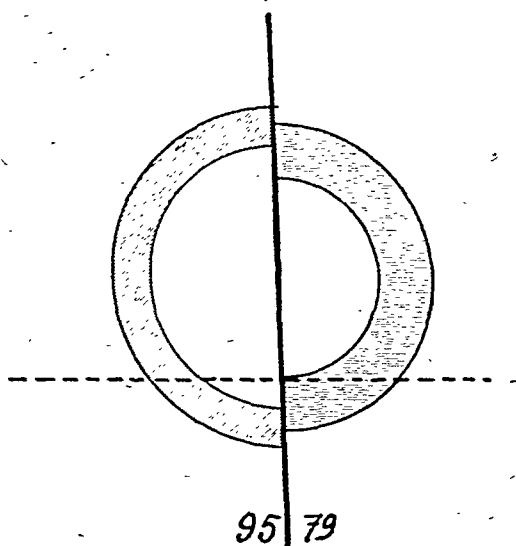


FIG. 9.

Fig. 9 represents the transverse sections 79 and 95, compared one with another. The dotted line shows how the longitudinal section of the optic nerve could cut only a septum of connective tissue in the cribriform lamina, and at the same time a nerve-bundle in the optic nerve. Fig. 10 represents it in a plastic way. Let us imagine the sections 79 and 95, made from a transparent material and lying one on another. The longitudinal section of the optic

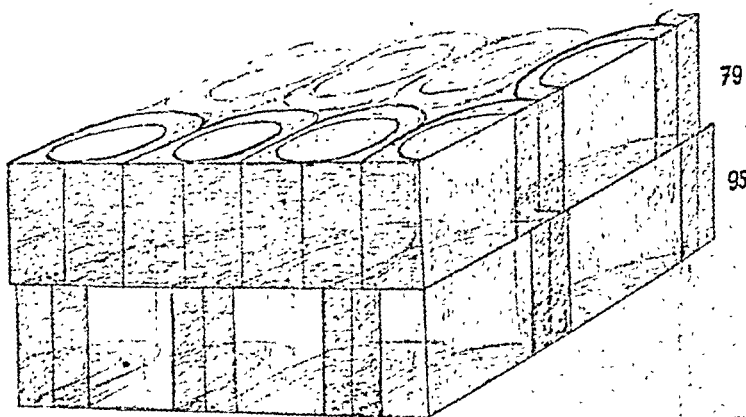


FIG 10

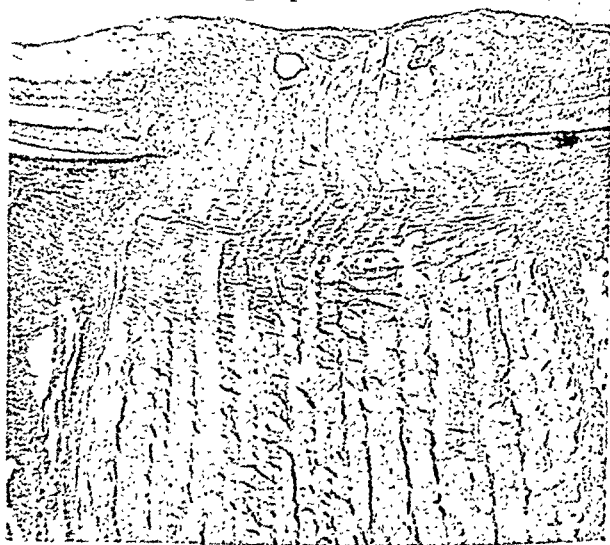


FIG 11

nerve passing the cribriform lamina would cut a septum of connective tissue, but passing the nerve itself would cut a nerve-bundle.

That is how "typical" histological pictures of the cribriform lamina can be explained. These pictures depend on the plane of the section. One can see diagrammatically in Fig. 9 that it is not

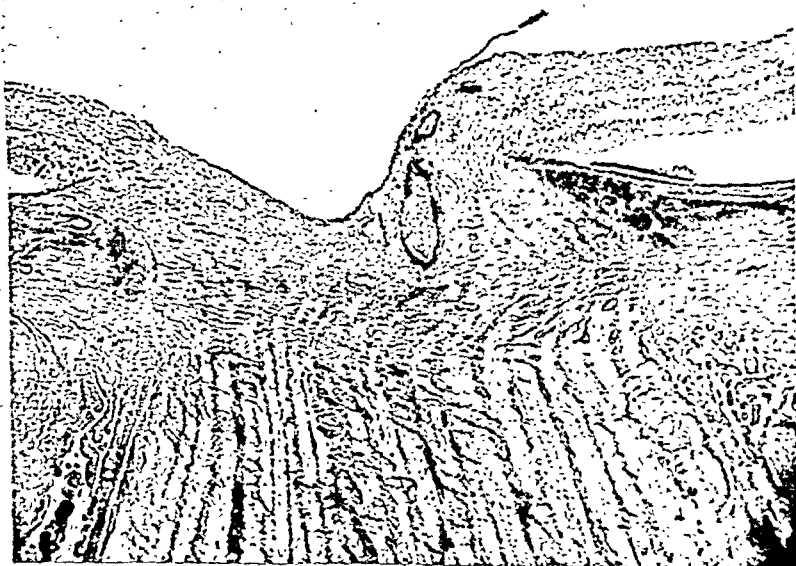


FIG. 12.

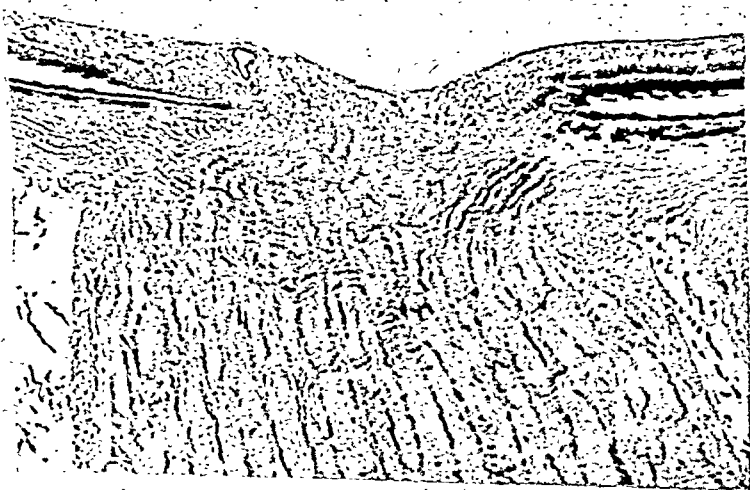


FIG. 13.

the greater thickness of septa of connective tissue in the cribriform lamina, but the greater diameter of myelinised nerve-fibres in the optic nerve that plays here a rôle. In Fig. 10 one may see the possibility of another cut which would pass nerve-bundles as well in the cribriform lamina as in the optic nerve. In such a case, in histological sections the pictures of the cribriform lamina would not be so typical. It is clear that in the same optic nerve one may obtain some pictures of strongly, and some of weakly, developed

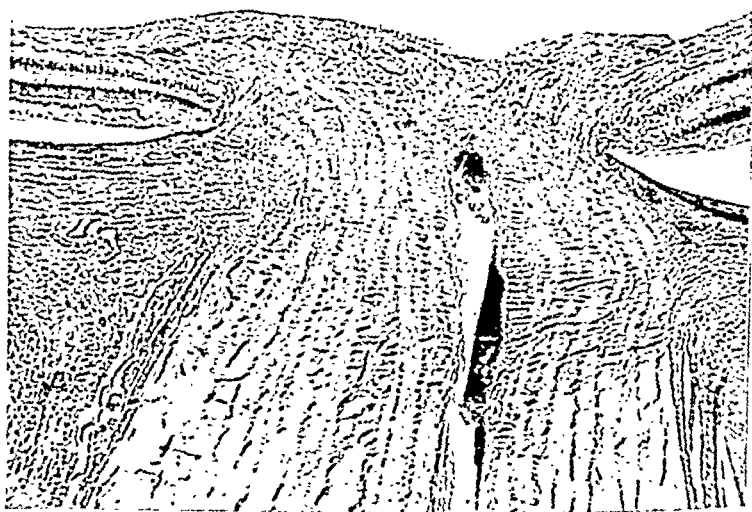


FIG. 14.

cribriform lamina according to the plane of cutting. By moving slightly the transparent cubes in Fig. 10 one could get the reversed pictures. A cut could have passed nerve-bundles in the cribriform lamina and septa of connective tissue in the optic nerve. In Figs. 11 and 12, the sharp back outline of the cribriform lamina is missing. Transverse fibres of connective tissue run across nerve-bundles in a similar way in the optic nerve as in the cribriform lamina. In Fig. 13, the lamina cribrosa seems to be composed of several levels. In Fig. 14 the cribriform lamina is hardly to be seen. Transverse fibres of septa run rather across the optic nerve.

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CONTACT LENSES *†

BY

IDA MANN

OXFORD

To us all here the subject of contact lenses is an interesting one and the foundation of a society for the scientific study of the problems connected with them needs no apology, but we are far from being a representative gathering. You will realise this when I tell you that only a short time ago an eminent scientist said to me "Why waste your time starting a contact lens society?" I replied "Why is it a waste of time?" and received the devastating answer, "Because everything is known about them. Optically, they provide a beautiful answer to many problems and practically they are useless, as no one can wear them for more than an hour and a half." I think we can say that this statement is largely untrue. In the first place, I hope to show in this address that we certainly do not know all about them and, secondly, I think no one here will dispute the fact that some people cannot even wear them for an hour and a half, while others notice no discomfort after ten times as long.

There would, therefore, seem to be good reason for a society for their study, even though so much has already been written on them and so many experiments performed. These experiments differ from the usual scientific experiment in that they have practically all had to be done on man. Animal experiment will not give us any of the answers we seek, since obviously we cannot test visual acuity in

* Received for publication, May 26, 1947.

† Presidential Address delivered to the Contact Lens Society.

an animal, nor can we enquire as to tolerance. Even gross experiments on corneal physiology are hardly applicable, the relative sizes of cornea and sclera differing between man and most other animals, as also does the anatomy of the limbus. We are, therefore, handicapped from the beginning by the introduction of innumerable variables (*e.g.*, of physiology, of pathology and of psychology) into each individual experiment and it is small wonder that, more than a hundred years after Thomas Young's original paper in the *Philosophical Transactions*, our knowledge is scanty and our chances of success in a given case still unpredictable. It should be the object of this society to formulate the problems which confront us and by the careful accumulation of accurate data to attempt to solve them.

It is obvious that our problems fall into two groups, those of optics and haptics. The former are not formidable and I do not propose to deal with them in detail, though you will all realise that much still remains to be done if we are to reach an optical accuracy comparable to that obtained with spectacle lenses. We have by no means mastered the problems of the introduction of prisms and of cylinders into contact lenses, nor are we very near the production of a bifocal contact lens. We are not even certain of the best material of which to make contact lenses, nor even whether that material yet exists. There is, therefore, room for collaboration with the physicist, the technician and the chemist.

The more pressing problems, however, are those concerned with tolerance. My own interest in the subject is purely clinical, and I have been ordering contact lenses more or less hopefully for the last ten years. Recently I realised that I knew very little about the results I had obtained and through the kindness of one of our secretaries, Mr. Cross, I was able to obtain a fair follow-up of 100 of my cases. This has been most illuminating, and has helped me to formulate some of our problems. All my cases were individually fitted, some of them after having tried Zeiss lenses with no success. In practically all of them contact lenses gave a better visual acuity than spectacles and were ordered for this reason. (I did not, in this follow-up, include cases with active pathological conditions necessitating contact lenses as part of treatment). The patients therefore had an incentive to persevere. Of the hundred patients, 61 were myopes, of whom 11 only wore -5.0 D.Sph. lens or under, the rest having very high corrections, up to -22 . The patients wore a single lens only, for monocular aphakia. Eleven had conical corneae and the remainder suffered from various disabilities, including high hypermetropia, binocular aphakia, corneal scars and dystrophies.

There were 46 males and 54 females and their ages ranged from 14 to 73 years. The majority were between 30 and 40 years old.

These patients were all asked whether, knowing what they now

know, they would go in for contact lenses again. Sixty-four replied unhesitatingly Yes, 19 gave an equally emphatic No, while 17 considered that certain improvements ought to be possible to make them comfortable and if these were done they would answer Yes. Can we discover any relationship between the tolerance and intolerance of these patients and either their ocular condition or their habits of wear? In the first place, must the lens be worn every day for tolerance to be good? Fifty-five patients wore their lenses every day and 47 of these had tolerance good enough to permit a full day's work in the lenses, 31 of them wearing them for more than 12 hours every day. The remaining eight (of the 55) had poor tolerance of less than six hours. On the other hand, nine patients who only wore their lenses occasionally had excellent tolerance also, so that regular wear is not proved to be essential. Most of the patients, however, who only wore their lenses occasionally had poor tolerance and gave this as a reason for not wearing them. Twenty-three of the 100 patients did not wear their lenses at all, but this was not always because of intolerance. In one case the patient's fingers were too rheumatic to manipulate the lens, in two cases a very dirty job was being done and they feared to put their dirty fingers near their eyes, in three others there was nervousness of the lens, but in many there was extreme intolerance.

Can we show that intolerance is associated with any special condition of the eye? If we consider the myopes we find that of the 61 questioned, 31 wore their lenses for more than eight hours every day, 16 wore them every day, but for less than eight hours, while 14 did not wear them at all and 10 of these 14 were high myopes who had much to gain visually. It does not therefore appear possible to relate tolerance to the presence or degree of myopia.

On the other hand, there does appear to be a correlation in the case of conical cornea, though the numbers are small. Of the 11 patients fitted, nine wore their lenses all day with success, and two could only wear them part of the day. None was a complete failure.

Monocular aphakia, though theoretically a good indication for contact lenses, does not in my series justify itself. Only two out of 10 patients wore their lenses at all and one of these has now given it up. The remaining eight all complained of diplopia.

Of the cases with various corneal conditions, only four or five had really good results, but this series is small and not homogeneous.

We therefore require more data on the type of condition suitable for contact lenses and it might well be that a correlation with tolerance is not possible.

Eighty-four of the patients replied, when asked whether they inserted their lenses dry or used a solution. Much has been written

and many theories advanced about the necessity for a solution and its ideal composition. The results in my series are surprising and show that much more work is needed and that theorising is unsatisfactory. Of these 84, 49 inserted their lenses dry and 23 of these had a tolerance of more than eight hours (often 14 to 16 hours); eight of the 49 had tolerance of four to eight hours and 10 of from two to four hours. Eight could not wear a lens at all. Twenty-two patients used normal saline. Seven of them had more than eight hours tolerance, eight of them four to eight hours, four of them two to four hours and three could not wear the lens.

The third group is, however, the most interesting. Thirteen patients had obviously experimented and with surprising results. One with 16 to 18 hours wear every day always puts his lens in his mouth before inserting it, one with eighteen hours used distilled water, as did another with very poor tolerance. One used horacic lotion and wore the lens six hours, nine used tap water and all had tolerance of eight to sixteen hours. We have obviously a lot to learn and unlearn about solutions.

What reasons were given for intolerance? In many the corneal veil was the most trouble and this was too variable for generalisation, coming on sometimes in half-an-hour, sometimes in 14 hours. In other cases pricking of the lids, smarting, grittiness, discharge, headache and many other symptoms were noted, and here we have our greatest problem. The problem of the veil is basically a physiological one and will probably be solved by some modification of fit, as is being shown by Dallos' experiments with holes and slits in the lens. But there are other problems connected with the state of the lids and the presence or absence of low grade infections. A whole series of experiments awaits an investigator here, many even applicable to animals. We have no idea of the alterations of the normal bacteriological flora of the eyes produced by contact lenses. Indeed, the surprising thing is the high local immunity of the conjunctiva and the rarity of serious infection. Low grade infection may be common; we do not know. It may account for sensations in the lids and for the accumulation of sticky secretion complained of by some patients.

So far, you will note, I have not touched on the question of fit at all and yet have raised a number of unanswered questions.

I will leave it to our next speaker to attempt to clarify our ideas on the problems of shape, size and fit, and will be content to close this very incomplete list of unanswered queries with a plea for the accumulation of more data and the planning of further experiments. I trust I have justified my contention that the scientific study of contact lenses is not a waste of time.

POST-OPERATIVE SECURITY IN CATARACT CASES

BY

T. G. WYNNE PARRY

BANGOR, N. WALES

THE simple procedure described below was devised to meet the case of an old lady of over eighty, whose mental and physical "wanderings" during her first night in hospital—where she had come for an extraction of one of her bilateral cataracts—gave rise to much foreboding as to her post-operative nursing.

The usual methods of trying to secure sound protection for the eye and the wound, were all thought of and discarded for various reasons: corneo-scleral suturing meant later removal of the suture, and did not seem to give the maximum security which one was aiming at; the van Lint flap had always appeared to me to give a line of tension across the middle of the cornea along the edge of the flap, and it seemed difficult with this method to get pressure to bear evenly on the wound, cornea, and sclera.

Finally, it was decided to try a complete conjunctival flap, *i.e.*, involving the whole circumference of the cornea. Accordingly, after the usual surface anaesthesia combined with retrobulbar injection anaesthesia and 7th nerve block over the mandible, a snip was made with scissors in the conjunctiva at the corneo-scleral margin to the outer side, a Weber's knife was inserted, and with the conjunctiva held taut with forceps at intervals all the way round, the conjunctiva was cut round the cornea. It was then undermined freely, and with 4/0 catgut, a purse string suture was run round the conjunctival edge—the two ends being at 5 and 7 o'clock. The conjunctival flap was now gently retracted away and a simple corneo-scleral section was made and, after iridectomy, the lens was extracted in capsule. The iris pillars were stroked back, and then, with the conjunctival margin helped over the wound with a couple of iris repositors, the purse string suture was drawn tight and tied gently but firmly. The whole cornea was thus covered with conjunctiva, and the general appearance gave one a most unusual feeling of security. The one eye only was bandaged, and the patient walked back to bed, and was ambulatory from the first day.

The suture started giving on the 3rd day, and the whole cornea was clear on the 14th—an unusually long period compared with succeeding cases. The whole convalescence was uneventful, the only treatment being my usual one of gtt. penicillin in flavazole, and gtt. atropine sulph. instilled every other day, and a daily inunction of ung. hydrarg.

Following this case, I carried out the same procedure in eleven

more cases. Out of this total of twelve cases done in the last 4 to 5 months—eight were intracapsular extractions, and four extracapsular. In one of the intracapsular cases there was some vitreous loss after the lens was extracted, but in neither this case nor any of the others were there any unusual symptoms, and the end results were excellent.

In the last eleven cases before the suture was finally drawn tight, gtt. penicillin and gtt. atropine were instilled into the mouth of "the bag." A small point which led to a little trouble with fixation of the eye in the first case, was that a small tag of conjunctiva should be left on the corneal margin at the point the operator usually prefers to place his fixation forceps—otherwise he finds it difficult to grip the eye without doing so on one of the recti muscles.

The suture was usually found to give about the 4th or 5th day, and the conjunctiva had retracted to the corneal margin by the 6th or 7th day. In only one case—for ultra-aesthetic reasons—was it necessary to help the conjunctiva back into place at one point on the 10th day.

The method is admittedly more tedious than the usual ones in vogue, but the feeling of complete security is very real. The purse string suture is easy to draw tightly and snugly, and the pressure over the whole cornea and wound is even and firm. The physiological conditions for wound healing are therefore ideal inasmuch as it is covered, supported, warm and sheltered from infection.

The fact that the patients can be ambulatory with safety after the first twenty-four hours is a marked advantage in these days of depleted nursing staffs, and any post-operative complications which may arise—with the exception of infection—are in effect dealt with before they happen.

I very much doubt if this procedure is original since nearly everything has been tried sometime, but I have not seen any reference to it in the literature available to me, and it is presented in the hope that it may be found to be a useful addition to the usual methods.

THE POWER OR "POWERS" OF A LENS

BY

JOSEPH I. PASCAL, M.D.

NEW YORK

If a doctor at an Ophthalmological Board examination were asked, "What is the power of the crystalline lens?", he might well ask which of the different "powers" of the lens was wanted. *In situ* the lens may be said to have three powers, based on actual focal

lengths, *i.e.* the principal power, the vertex power, and the apex power. Placed in air it is very much more powerful, and again may be said to have three powers, the principal power, the vertex power, and the apex power. But in addition, and this is the most important point, *in situ* the lens has also a "reduced" power. This is the power as determined by the reduced focal length. This latter power is the real significant power and the one used by Gullstrand in his calculations of the dioptric power of the eye.

The definition of surface power and lens power, following the lead of Gullstrand, is based upon "reduced focal length". The power in dioptres is the reciprocal of the reduced focal length. The two actual focal lengths of any optical system are equal, when the first medium (or its index of refraction) is the same as the last medium (or same index of refraction). The two focal lengths are unequal when the first and last media are different. But in either case, there is only one reduced focal length. The refractive power of the system can be expressed by a single value, the reciprocal of this focal length, even when there are two actual focal lengths as in the case of the cornea, or the eye as a whole.

The reduced focal length is simply defined as the actual focal length divided by the corresponding index of refraction. Thus if f_1 and f_2 express the two actual focal lengths of a system, the reduced focal length $f(r)$ is equal to f_1 divided by n_1 or f_2 divided by n_2 . The whole subject of "reduced" distance, "reduced" light vergence, "reduced" focal length, etc. is something that is basic and merits the study of every ophthalmologist. Unfortunately, the subject is hardly touched upon in the standard books on ophthalmology. It is true that for a lens in air, the reduced focal length is equal to the actual focal length and its significance and use may be ignored. But when studying the dioptries of the cornea or of the eye as a whole where the reduced focal length is not equal to the actual focal length, a knowledge of this subject is essential. Books which do not point out the difference between the two systems create for the student-reader unnecessary difficulties and sometimes confusion.

For example, in Duke-Elder's Text-book of Ophthalmology, vol. I, page 748, there are tables contrasting the finding of Tscherning with those of Gullstrand relative to the position of the cardinal points, the dioptric powers of the components, etc. of the schematic eye. The table gives Tscherning's figure for the refractive power of the crystalline lens (*in situ*) as 16.01 D. and Gullstrand's as 19.11 D. This apparently means that Gullstrand using different data for his calculations found the power of the crystalline lens to be 3.10 dioptres more than what Tscherning found. But there is no mention of the fact that Tscherning used "actual" focal length for his power determination and Gullstrand used "reduced" focal length. In

fact, if the lens power as found by Tscherning be changed to "reduced" power it comes out even more than Gullstrand's, being 21.40 D. That is, the reduced power of the crystalline lens, using Tscherning's data for radii, indices and thickness is more by 2.29 D. than the value found by Gullstrand.*

The method of calculating reduced surface power directly is very much simplified by what has been called the "dam" formula, $D = aM$. Here D stands for dioptries of power (reciprocal of "reduced" focal length) "a" stands for the difference between the two indices, (i.e. "a" amount 2nd index is above 1st index) and M stands for curvature in metres. The latter is obtained by dividing the radius into 100, or 1,000 or 40 depending upon whether the radius is given in cm. in mm. or in inches.

As an illustration we may apply this formula to finding the "reduced" power of the crystalline lens, using Tscherning's data which are as follows: $r_1 = 10.2 \text{ mm.}$, $r_2 = 6.22 \text{ mm.}$ $t = 4.06 \text{ mm.}$ n of lens = 1.42, and n of aqueous — vitreous equals 1.3365.

For first surface $D = (1.42 - 1.3365) \times \frac{1,000}{10.2}$; this works out to 8.19.

For second surface $D = (1.3365 - 1.42) \times \frac{1,000}{-6.22}$; this works out to 13.42.

The sum of the two surface powers is 21.61 D. The effect of thickness reduces the total power by 0.21, giving a power of 21.40 D.

RESTORATION OF THE CHAMBER AFTER INTRA-CAPSULAR EXTRACTION

BY

Prof. A. KETTESY

DEBRECEN, HUNGARY

It is well-known, since doing intra-capsular cataract extraction that we see delayed restoration of the anterior chamber more frequently than before.

This complication in the after-treatment has generally no significance at all, more properly, less significance than in the extra-capsular extraction, when there is the possibility of capsular or cortical particles left in the section. Nor has it any significance after the intra-capsular extraction either, if a shallow chamber is present if for only a short time. The well-known and easily

* The illustration taken from Duke-Elder's work is not meant as a criticism, but just to point out this omission even in the most scholarly and best known book on the subject.

verified causes of the temporary and periodic disappearance of the anterior chamber are delayed healing, re-opening of the wound, and choroidal detachment. The eye is soft, which is a re-assuring symptom.

But it happens also—luckily very seldom—that the chamber does not form at all; this very serious sign seems not to be appreciated in detail. The anterior chamber, after a smooth intra-capsular operation, without any discoverable cause, does not reform, leading eventually to the loss of the eye. The wound is well co-apted and closed, the eye is quiet, the palpated tension is normal, Seidel's test is negative, and there is no sign of glaucoma for the first 12-14 days. Then a fine haziness of the cornea appears as the first sign of glaucoma, the tension slowly rises to 30-40 Hg/mm. and without adequate medical aid the eye becomes blind.

In the last 20 years I have lost 2 eyes in this way, in spite of having performed in the second case posterior sclerotomy, cyclo-dialysis and trephining. The chamber could not be restored. (As I do an average of 250 cataract operations a year, including about 200 intra-capsular extractions, this makes together with the case described below 0.7 per cent. of the intra-capsulars for the last 20 years).

It was by chance that a third case showed the way to restore the anterior chamber and gave also a possible explanation. Intra-capsular extraction was performed on both eyes of an old lady. It was a straightforward case, without any sign of impending trouble. The operation was uneventful in both eyes with my routine procedure: Knapp-Stanculeanu-Török, iridectomy, conjunctival hooding of the wound, akinesia and retrobulbar injection. We instil as a rule pilocarpine after intra-capsular extraction.

No restoration of the chamber occurred in either eye. There was no irritation, the wound closed, and no hypotony. We waited 6 days for spontaneous restoration.

On the seventh day we started to do something for the restoration of the chamber. We instilled 8 per cent. pilocarpine together with cocaine and adrenalin, in order to raise the tone of sphincter and dilator simultaneously, a proceeding that leads sometimes to success. But in this case it failed, as well as repeating it with 1 per cent. adrenalin the next day.

On the ninth and tenth days we joined to the instillations intravenous injections of 40 per cent. dextrose in the hope of obtaining a result by dehydrating the vitreous, but in vain. Dionine powder into the conjunctival sac and sub-conjunctival injection of saline solution proved to be similarly inefficacious.

On the twelfth day after the operation the loss of both eyes seemed to be imminent, so we started operative interventions. Both wounds seemed to be well closed, palpated tension was normal, no lacrimation, vision good:

In remembrance of the uselessness of posterior sclerotomy in a similar case, I decided to do cyclodialysis on both sides, after the modification of Blaskovic's (*a tergo*) carried out in front of the external rectus.

The end of Heine's spatula, forcing its way during the operation into the chamber between iris and cornea, got into the pupillary area by mistake, and injured the hyaloidea. The vitreous streamed immediately forth, pushed the iris back and filled the chamber. The chamber was restored (of course a vitreous-filled chamber). Eye-surgeons see the same thing occasionally, when, during cataract extractions the hyaloidea is broken and vitreous fills the anterior chamber.

On the fellow-eye I brought about the same event on purpose. I pushed the point of the spatula into the pupillary area and broke the hyaloidea by a little movement sideways and backwards. The result was the same, the chamber was immediately restored.

The further course of the case was satisfactory. The palpated tension of the eye remained normal, taken by tonometer 6 days later it was 22 Hg/mm. Corrected vision 6/9 and 6/12. Slit-lamp examination showed the well-known signs of the vitreous-filled chamber: in the aqueous there were waving from the pupil ramifying fibres of the vitreous.

Since then I had no opportunity to perform this operation, that could be called restoration of the anterior chamber. It was only considered once, but the chamber restored itself spontaneously on the eleventh day.

The plain vitreous chamber has, as far as our experience reaches, scarcely any significance regarding post-operative glaucoma. It is true, text-books mention the loss of vitreous and the absence of the hyaloidea amongst the causes of post-operative glaucoma, but there are also plenty of observations to the contrary. Yet, admitting the possibility of bringing about the danger of later glaucoma, this is a small drawback in comparison to the direct and rapid loss of an eye after the operation.

It is interesting to note that the whole vast literature of cataract-extraction does not mention the primary, nor glaucomatous non-reappearance of the chamber, only the prolonged restoration. Of course, it must have been very rare in the pré-intracapsular period, and whenever it occurred, it was traced back to non-closure of the wound, to epithelial immigration and chiefly to an unobserved glaucomatous tendency, promoted by the operation. Though the latter possibility cannot be rejected with absolute sureness, I am convinced, in my cases there was no primary glaucomatous disposition. It seems to me more probable, the cause was purely mechanical. The iris was flat, adhering to the cornea, the pupil

was tightly filled with the inelastic hyaloidea, capillary adhesion closed up the chamber completely. Under such conditions spontaneous reversibility is hardly imaginable. The retained aqueous must be taken up by the vitreous, its swelling presses iris and hyaloidea the more to the cornea: a vicious circle thus arises which can be interrupted only by operative intervention. A similar situation is well known, of course only after glaucoma operations when it is the lens, that obturates the chamber. There is also an account of trying to restore the chamber in such cases. Asayama and Chikakiyo blow air between iris and cornea if after Elliot's trephining the chamber will not reform (*Zentralblatt f. Ophthalm.*, Vol. XLII, p. 599).

ANNOTATIONS

Neuro-paralytic Keratitis

Although alcohol injection of the Gasserian ganglion is a strikingly successful form of treatment for trigeminal neuralgia, the patients who submit to it are always haunted by the possibility of developing lesions in the anaesthetic cornea which, if untreated, may lead to serious consequences involving, sometimes, corneal perforation, and loss of the eye. The cause of the condition has been a subject of discussion among physiologists for many years, one group maintaining that the cornea has trophic nerve fibres which in some mysterious way look after its nutrition, and that when these fibres are blocked or severed, the cornea suffers; the other group maintains that anaesthesia of the cornea is sufficient reason for its breakdown, minute traumata occurring, unnoticed by the patient but leading to subsequent keratitis. This has always seemed to us an unsatisfactory explanation, in view of the fact that the earliest observable change in the cornea consists of the development of numerous pinpoint areas in the exposed part which stain with fluorescein, and it is difficult to imagine any form of trauma which would produce this.

The only method of treatment until recently has been to provide some sort of cover for the cornea. The ideal cover is, of course, the lids, and for many years now the operation of tarsorrhaphy has held the field, with resulting inability to use the eye. This disadvantage can be mitigated by gradually dividing the adhesions once the cornea has regained its normal state, and it is frequently found that an adhesion only 3 mm. wide lying over the outer limbus is enough to maintain integrity of the corneal epithelium. Even this, however, constitutes a fairly conspicuous disfigurement, so other methods have been sought for. The application of a pad is dangerous

because the patient may unconsciously open the eye underneath it and inflict severe trauma on the cornea.

A method described by Klein consists in the use of a plastic contact lens. He claimed several successful results, but there is always the possibility of the patient injuring the anaesthetic eye when inserting the contact lens, and perhaps not getting it into the correct position. We have known of one case in which the patient allowed the lower ledge of the lens to project over the lower lid. If an extrinsic protection is to be provided it should take the form of a rigid and preferably transparent shield, with sufficient bulge on it to stand well away from the eye and not come into contact with it, even if the patient lies on the shield during sleep. If facial paralysis is also present, as sometimes happens in other neurological conditions, then it must be air tight so that moisture is retained, but this is not necessary in ordinary trigeminal cases when sensory denervation alone has to be considered.

One hopes that these methods of treatment will soon become matters of historical interest rather than current procedures. Many years ago Mackenzie of Toronto described the effect of sympathectomy in causing healing of ulcers of the face after trigeminal denervation, and Norman Dott has found that it causes rapid healing of trophic corneal, nasal or other ulcerations of the skin dependent on trigeminal denervation. The effect lasts for many years, possibly indefinitely, and he is now in the habit of doing a prophylactic sympathectomy before or after alcohol injection of the ganglion. None of the cases treated in this way has so far ulcerated.

This procedure is not, of course, required in cases of trigeminal neuralgia where the first division is not involved because in them, by fractional section of the sensory root of the 5th nerve, it is possible to retain sensation in the eye and lids.

On observation

We do not hesitate to affirm that good powers of observation are among the most important items in the outfit of any member of the medical profession. Students should be taught to cultivate the faculty from the start of their careers and the practice should be kept up and not dropped as soon as the qualifying exams have been surmounted, for no one will deny that in our profession we remain students all our lives. The youth of the writer's generation had the importance of observation powerfully instilled into their minds by those admirable Sherlock Holmes stories of the late Sir Arthur Conan Doyle. Holmes was never at fault in his observations and Sir Arthur was a born story teller. One of Holmes' remarks that has always stuck in our mind was to the effect that a man's brain was like a room of limited capacity, and that the wise man only took

into it information that was likely to be of use to him in his work and did not get it cluttered up with odds and ends, which, however interesting in themselves, would crowd out more useful facts. John Hunter must have meant something of the sort when he declared, after coming down from Oxford, that they had tried to stuff him with Latin and Greek and make an old woman of him.

Of those ophthalmic surgeons we have known we should place the late Mr. William Lang as nearest the Holmes ideal. Mr. Lang was phenomenally observant and never seemed to miss anything. A friend of ours used to declare that in his early days in ophthalmology he made a habit of accompanying a clinical assistant to the hospital each day, and they used to award marks to each other for any ocular condition they observed in people in their walk. Gross cases of squint would be marked at the figure 2, less marked strabismus might count 5, an empty socket 1, but a well fitting glass eye 10 and so on.

There is something to be said for the idea and it need not be confined to eye conditions. A person who limps in his gait may have several possible conditions, including corns to account for it. Even in reading a book there are opportunities for cultivating mental observation. We wonder if the average reader of *Pickwick* has noted the fact that on Mr. Peter Magnus's first appearance he was wearing blue spectacles and, that when he was introducing Miss Witherfield to Mr. Pickwick next day, the spectacles had changed to green? Even the worthy Dr. Watson suffered from an Afghan bullet in his shoulder in one work of fiction by Doyle and in his leg in another.

BOOK NOTICES

Eye Manifestations of Internal Diseases. By I. S. TASSMAN. Second edition. 614 pp., 243 illustrations, including 24 in colour. Published by Henry Kimpton, London. 1946. Price, 50/-

Many ophthalmologists will be pleased to see the second edition of Tassman's book on the ocular manifestations of general diseases. It will be remembered that the book is intended to meet the needs both of the ophthalmologist and the general physician; and it is indeed extraordinary how much of ophthalmology can be included within this scope. After an introductory part describing the normal structure of the eye and its routine examination in a simple manner to suit those not experienced in ophthalmology, two chapters follow dealing cursorily with structural abnormalities and congenital and hereditary manifestations in the eye. Then follow chapters on the ocular complications of general infections of all types including bacterial, virus, fungus and parasitic infections, intoxications due to

drugs and chemicals, cardiovascular, haematogenous, metabolic and endocrine diseases, avitaminosis and disturbances of nutrition (which, surprisingly perhaps, include botulism), central nervous diseases, diseases of the skin, and of the bones of the skull. It is obvious that the scope of the book is very large, embracing practically the whole of ophthalmology, and its value as a source of reference is considerable, the emphasis being placed throughout on the ocular manifestations of the disease and not on a description of the general aspects of the disease itself. The text has been brought considerably up-to-date in this edition and several new additions appear—ocular allergy, epidemic kerato-conjunctivitis, Hurler's disease, lymphogranuloma venereum, Bowen's disease and toxoplasmosis, to mention a few. The greatest deficiency of the book is perhaps the lack of sequence and correlation between different conditions. Thus epidemic kerato-conjunctivitis is treated as a new specific entity without any correlation with other related conditions such as superficial punctate or nummular keratitis while the common development of disciform keratitis from virus infections is unstressed. On the whole, however, the book is a valuable addition to any ophthalmological library.

Diseases of Children's Eyes: By JAMES HAMILTON DOGGART.
288 pages with 210 illustrations including 32 coloured plates.
Henry Kimpton, London. 1947. 42/-

A book devoted entirely to the eye diseases of children is unusual. It is, however, by no means out of place for there is much in ophthalmology, as indeed in the whole of medicine, that is peculiar to the young. The methods of examination are to a large extent specialised with their necessary dependence on objective testing; to some extent the anatomy is specialised (as witness the development of the air sinuses); congenital deformities—a common and striking aspect of ocular pathology—are prominent in the problems involved; certain diseases are peculiar to childhood, and many problems—particularly those of squint—assume unusual importance in this stage of life. It is true that to some extent the aspects of some of these problems as applied to children lose their perspective in many text-books and courses of systematic teaching, but it is more important that their urgency is often forgotten by the family physician and sometimes by the paediatrician. In so far as it will tend to emphasize this aspect of ophthalmology, Doggart's book is welcome and useful.

The book itself is comprehensive. It opens with an outline of the general principles of examination and a discussion, occupying four chapters, of the anatomy of the eye and its adnexa. It may be questioned whether in a book of this type so much elaboration of this section is necessary; the student of ophthalmology will get it elsewhere, and the physician may be overburdened with detail. The

next four chapters deal comprehensively and well with the development of the eye and its adnexa and the congenital abnormalities which affect these structures. There follow chapters on the ocular complications and sequelae of systemic diseases in childhood, methods of treatment and the principles of ocular welfare in the young—special schools, lighting conditions and ocular hygiene generally. The chapter on refraction contains sound advice on the inadvisability of debarring myopic children from reasonable educational development. There are five chapters on disorders of ocular motility (heterophoria, convergence defects, squint, nystagmus, etc.) and the remainder of the book concerns itself with a brief description of the commoner diseases of the various structures of the eye, the orbit and the lacrimal apparatus.

The author has had a long experience of ophthalmic problems in the young through his association with the Children's Hospital, Great Ormond Street, London; and the book bears ample evidence of his sound appreciation of the problems involved. There are few faults in detail—recent research, for example, is not represented in the delineation of the pupillary paths—and its general philosophy and teaching, although some of the opinions expressed are personal, are of great interest and value. The book is beautifully produced despite the difficulties of the time and amply illustrated. Both publisher and author are to be congratulated.

A Treatise on Gonioscopy. By MANUEL URIBE TRONCOSO. P. 306. 117 illustrations, 35 in colour. Published by F. A. Davis, Philadelphia. 1949.

Although Trantas in 1892 succeeded in examining the angle of the anterior chamber with the ophthalmoscope by employing finger pressure over the outer part of the limbus in cases wherein it was especially deep, and Mizuo in 1914 obtained a distorted view of the lower part by filling the inferior fornix with water, the first adequate examinations were made by Salzmann in 1914 who used a contact glass and direct ophthalmoscopy and Koeppe who used it in combination with the slit-lamp. These pioneers laid the foundation of our clinical knowledge of the vagaries of the angle, but it was not until Troncoso of New York introduced his gonioscope in 1925—a microscope combined with a periscope—that this knowledge became exact and widespread and of clinical value. Previously the state and affections of the angle were interpreted in terms of microscopic sections, and many of the deductions as applied to the living eye therefrom have been shown to be erroneous. To-day a new branch of ophthalmic diagnosis has developed and become popularized, and sufficient observations have accumulated to establish its value and define its potentialities. It is opportune, therefore, that this knowledge should now be gathered together and summarized, and it

is fitting that the book should come from the pen of Uribe Troncoso to whose enthusiasm and immense capacity for patient and accurate observation gonioscopy owes its place in ophthalmology to-day.

Troncoso's book is a delightful one—well written, easy to read and full of interest. The first chapters are devoted to a description of the comparative anatomy of the angle in man, the comparative physiology of the outflow of the aqueous, and (written by Ida Mann) the embryological development of this region. There follow the story of the development of gonioscopy and a very clear and adequate description of the various techniques which have been used, a description of the gonioscopic appearances of the normal angle, and finally its abnormal appearances in senility, in congenital deformities, in injuries, inflammations, tumours, glaucoma (in great detail in three chapters), in post-operative glaucomatous states and in various miscellaneous diseases. It is in glaucoma, of course, that the method has excited the greatest interest and is of the most clinical value: from the point of view of pathology it has demonstrated that peripheral synechiæ are the result, and not the cause of raised intra-ocular pressure, and has laid the foundation for a division (now becoming popular) of the disease into a wide-angle and narrow-angle type. From the clinical point of view the greatest value of the method is to suggest lines of appropriate treatment and to observe the efficacy of operation.

So clearly is the book written that it would be easy for the tyro to undertake gonioscopy with it alone as a guide. To a considerable extent this is rendered possible by the number and excellence of the illustrations, and further study is facilitated by the full bibliographies. Obviously Troncoso has enjoyed his life-work; his writing shows his enthusiasm and it will surely inspire others to profit by his experience, and advance still further a fascinating diagnostic technique.

NOTES

National Society for the Prevention of Blindness, Inc. Poster

AN attractive poster captioned "Seeing Through Life" is available from the National Society for the Prevention of Blindness at 1790 Broadway, New York 19, New York. Recently revised and reprinted in bright colours, the poster emphasizes the measures that conserve and protect eyesight from the pre-natal stage through old age. It is suitable for use in connection with health and safety education programmes. Size, 19×25; price, 30 cents per single copy, with reductions on quantity orders. If possible, remittance should accompany order.

* * * *

Corrigendum

IN the article on familial cataract, p. 391, the last word of the legend beneath fig. 7 should read extraction, not extension.

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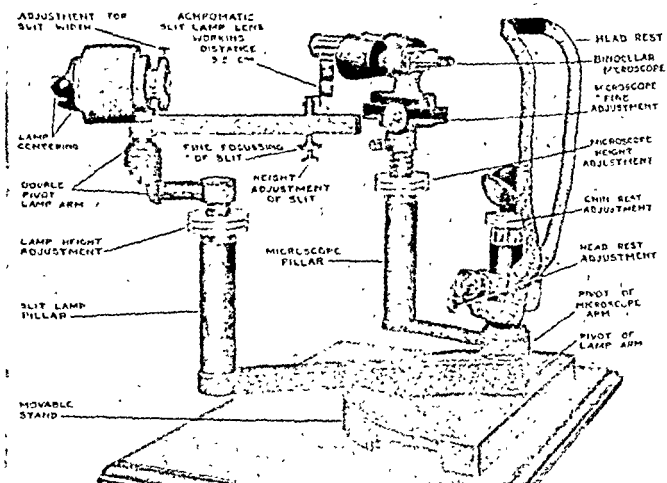
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THE BRITISH JOURNAL OF OPHTHALMOLOGY

OCTOBER, 1947

COMMUNICATIONS

THE TREATMENT OF TRAUMATIC DIPLOPIA

BY

Group Captain J. C. NEELY

CONSULTANT IN OPHTHALMOLOGY, ROYAL AIR FORCE

Scope of the work

In the Royal Air Force in 1937 an investigation was carried out concerning the incidence and the causes of accommodative asthenopia.

Before the war motor cycle accidents rather than aircraft crashes were responsible for the majority of head injuries, and they, in their turn, were often the cause of accommodative asthenopia. Usually some weakness of convergence was found to be associated with the condition, although, as the adverb implies, this association was by no means invariable. The investigation was therefore carried a stage further, and the other extra-ocular muscles were examined.

To make a finer estimate of the paresis of the particular muscle or muscles at fault, the Hess screen was used to record the initial weakness and the stages of any subsequent recovery. The Hess charts 1 and 2 are examples which show the extent of the diplopia when an airman was first examined following an aeroplane accident, and at a later date, when recovery was still taking place.

Owing to the author's service overseas, this investigation was not completed; it is mentioned, however, in order to support the contention that cases of traumatic diplopia, although uncommon in times of peace, are not by any means rare. From experience of this war, it seems that the condition is more common than is realised, not only by doctors, but also by ophthalmologists.

On returning to England in 1942, a fresh start was made to gather together the threads of this investigation and to watch the pattern emerge on the fuller canvas afforded by the material of war. At the beginning of the 1939-45 war, Lyle, who was then serving in the R.A.F., was working on the composition of this tangled tapestry, and by 1942 the form of the picture may be said to have taken definite shape. The material of this paper has been drawn from the records of patients, who were seen by the writer at a Military Hospital for Head Injuries, and at a R.A.F. Hospital. In each case, the description includes that of the injury which caused the diplopia, the ocular state of the patient before and after treatment, and the final disposal. The investigation of the case and the treatment, both surgical and orthoptic, now follow established principles. Under the more exacting conditions of war, the results have been tested and, because symptoms have been relieved and in the majority of cases the patient has returned to duty, these results are thought to show a definite advance in modern practice.

Previous history

Looking back through the history of ophthalmology for those oculists of the past who first thought of the various ways by which crooked eyes might be straightened and diplopia overcome, it seems that to Chevalier Taylor, that prince of ophthalmic charlatans, the credit must be given of being the first to think of curing squints by operations.¹ His operative technique is described by Lecat² and is of interest in showing how near Taylor was in theory to modern practice and also in his explanation of the trick which he used to straighten the squinting eye. Lecat, in writing of Taylor's visit to Rouen in 1743, tells how the door of his hotel was guarded by soldiers, and that an appointment had to be made in order to see him. He then describes his operations, and comes to "*La grande operation, le plus merveilleuse de toutes*"; this was the operation for the cure of squint. Taylor picked up a piece of conjunctiva at the inferior part of the globe, cut it off with scissors, applied a bandage to the sound eye, and the miraculous occurred; the squinting eye became straight. Taylor explained that the reason for the operation was to cut a nerve filament to the overacting muscle. Coats³ considers that Taylor became

convinced that the operation was impracticable and that, in order not to lose the dishonest emoluments, he devised the fraudulent procedure described by Lecat. How Lecat exposed him at a dinner party by means of a dissected human head is of interest, but not germane to the subject; it is pertinent to note, however, that Taylor knew of a man who was greatly troubled with traumatic diplopia, that he clearly distinguished between ordinary squint and paralytic squint due to injury of the ocular muscles, and knew also of the development of suppression in the squinting eye. After Taylor's "Operation," "The proposal to divide a muscle for a squint slumbered for nearly a century till it was put into practice on the cadaver by Strohmeyer, and on the living in 1838 by Dieffenbach."

Dieffenbach was the first to carry out the surgical procedure of exposing the belly of the overacting muscle and cutting across its fibres: the operation had to be abandoned, however, because the muscle lost all its power.⁴ Since that time innumerable operations for squint have been devised; they nearly all concern themselves with the problem of straightening a lateral deviation. Compared with these, there are very few references in the scientific press to the vertical deviations and their correction; those relating to traumatic diplopia and its treatment are even more scanty, and the librarian of the Royal Society of Medicine could provide only two⁵ and ⁶. In 1864 von Graefe was investigating the causes of ocular torticollis, and it was treated by operation by Knapp in 1874. The technique of operating on the inferior oblique of the same side in cases of paralysis of the superior oblique was developed by Landolt, while von Graefe and von Kries were more in favour of a tenotomy of the inferior rectus of the opposite side.⁷ Stanculeanu⁸ describes three cases of vertical diplopia: two following radical cure of the frontal sinus, and one due to an injury by a cow's horn. In all these subjects the operation performed was an advancement of the inferior rectus on the side of the injury. The merits and shortcomings of this operation will be discussed later. De Morsier at Barbey⁹ in describing the predominance of unilateral provoked nystagmus in cases of post concussional giddiness included that of a farmer who suffered from a traumatic diplopia due to paralysis of the right superior oblique. No treatment, however, was included in the account which was of the nystagmus rather than the diplopia.

Present day practice

It may be seen from the paucity of historical material that the condition has been treated only by a very few ophthalmic surgeons in the past, and that no wide study of the possibilities of treatment or of the results of operation has been made until the recent war.

The attitude of the majority of British ophthalmologists appears to be but a continuation of this policy, and the impression is widespread that little can be done when dealing with a case of traumatic diplopia. Prisms are sometimes prescribed if the ocular deviation is not too marked, or occlusion of the affected eye may be advised until suppression of the false image makes life more endurable for the sufferer. Operative measures are hardly ever advocated.

It seems that the following extracts from two popular text-books of ophthalmology may be quoted as reflecting present day thought and teaching. "The diplopia may sometimes be relieved by suitable prisms, but this treatment is rarely of much use, owing to the variation in the amount of deviation in different positions of the eyes. Occasionally good is done by exercising the weak muscle with strong prisms. In old cases an operation may be indicated, usually a tenotomy of the antagonist with the advancement of the paralysed muscle, thus putting the affected muscle under better mechanical conditions. It is only suitable for parietic, not paralytic, cases and should *never* be adopted until all other means fail. It is, therefore, seldom indicated."¹⁰ "Paralysis of the vertical recti, or an oblique muscle, is much more difficult to relieve, and the last has been, for the most part, regarded as irremediable by operation."¹⁴ Treatment by prisms of the required strength divided between the two eyes has been the method of choice of most British ophthalmologists. This method is rarely satisfactory, and renders the wearer completely dependent on his glasses. In civilian life this is a great disadvantage, especially when the subject is playing games or driving a car, while in a flying service it effectively bars the wearer from many active appointments.

It may be seen that the somewhat ill marked sign-posts which were erected on the Continent towards the end of the last century became practically indecipherable, and were too few in number to be of use in directing ophthalmologists towards a proven way of treatment. Of this generation it seems that it was Chavasse¹¹ who again began to show the way. He established certain principles regarding the phenomenon of overaction of the synergic muscle, secondary deviation of the sound eye, and the tendency that the incomitance has to disappear with the passage of time. Before his death, he also succeeded in applying the principles of orthopaedic surgery—advocated by Haab in 1905—to the correction of defects of the ocular muscles.¹² It is these principles, enthusiastically taken up by Keith Lyle, coupled with the use of orthoptic exercises, that has made it possible to treat successfully the numerous cases of diplopia caused by the head injuries of the war.

The occurrence of diplopia

In peace time cases of traumatic diplopia are undoubtedly rare. In the Royal Air Force, where high spirited and not very prudent young men are apt to travel fast in aeroplanes or on motor cycles, the opportunities for acquiring a head injury might be expected to be greater than among the less mobile members of the community. Even so, not more than five or six airmen suffering from traumatic diplopia were seen in 1937-8.

It seems, therefore, that one of the reasons why the surgery of the vertical and oblique muscles of the eye has remained so undeveloped is the lack of opportunity most eye specialists have had for the diagnosis and treatment of traumatic diplopia; having little experience of the condition, and accepting the popular opinion that nothing can be done in these cases, they content themselves with prescribing prisms, or encouraging suppression of the false image by means of a frosted glass. How the war of 1914-18 did not lead to a review of the existing methods of treatment is not fully understood, for the heavy casualties from the western front must have included a great number of men suffering from head injuries, and many of them must have had double vision.

Ophthalmic services were not organised, however, to the same extent as in the recent war, and in France a great deal of eye surgery was done by general surgeons.

During the year 1942 to mid 1944 more than 200 cases of diplopia due to injury were seen at routine examinations in two service hospitals. It is not possible to say what percentage of the total head injuries this figure represents. Many men were wounded so seriously that they could not be examined and a number were transferred to other hospitals as soon as their general condition had sufficiently improved.

On the other hand, patients suffering from diplopia caused by head injuries received some months before (cases from overseas, etc.) were often referred to hospital for treatment.

A few cases were seen when they were sent for an opinion, on recurrence of symptoms after they had previously been passed fit for flying elsewhere. One pilot said that when he started to fly again, after a crash, he saw two runways on coming in to land, and in his confusion stalled an Oxford from 20 feet, and crashed again.

When discussing the incidence of traumatic diplopia it should be mentioned that it is often present in cases of trauma to the head and face, but this is frequently masked by other injuries, especially burns, or it may develop later owing to an injury to the orbit which forces a particular muscle to work at a mechanical disadvantage. Under these conditions, merely to maintain the eyes in their

primary positions will cause some discomfort and fatigue; moving the eyes in the direction of action of the paresed muscle will increase the fatigue until the "fusion hold" is strained to breaking point, and the object looked at splits in two.

Writing on this power of fusion, Bielschowsky¹³ remarks that "the stronger the fusion mechanism, the longer and more easily it will keep the tendency to dissociated vertical movements latent." This is borne out by the history of many patients, particularly that of an R.A.F. policeman. This man suffered from 16 pd of right hyperphoria, but his power of fusion was such that he did not see double unless he went to the cinema. At his last visit he found he had to force his head further and further back, until eventually he had to shut one eye to overcome the diplopia. After one operation and orthoptic exercises, the hyperphoria was reduced to 8 pd. His eyes remained so comfortable and his power of fusion so strong that he considered further operation unnecessary.

The nature of diplopia

Traumatic diplopia may be divided into three categories:—

1. Diplopia caused by the alteration in the "seating" of the eye, and the mechanical embarrassment under which certain movements have to be made.
2. Diplopia due to injury of the muscles themselves by direct or indirect violence.
3. Diplopia due to a paralysis of an extra-ocular muscle, following damage to its motor nerve by intra-cranial injury.

Group 1 comprises those cases of facial fracture illustrated by case notes 1 to 9. These often result from aircraft accidents. A pilot tends to strike his head on the gunsight when he crashes, and fractures the middle third of his face, or the zygomatic malar complex.

In considering Group 2, direct injury to a particular muscle is a somewhat rare occurrence. It sometimes follows a frontal sinus operation or perforating injuries below the eye. (Cases 11, 13 and 16.)

Indirect violence is more difficult definitely to prove, but it is reasonable to suppose that the eye, suspended in the orbit in its cone of muscles, must suffer displacement when the skull receives a heavy blow. Ridley¹⁴ puts forward the suggestion that the optic nerve is injured in this way through the eye-ball being "left behind" when the head is struck. It is thought that in a similar manner the eye muscles are contused, and diplopia, from the resulting paresis, is produced. The rapidity with which many of these cases recover lends colour to the view that there has been a minor injury to the muscle without coincident damage to the cranial nerve supply.

The extra-ocular muscles

Before any specific treatment can be undertaken, it is necessary as in other medical and surgical conditions, to make an accurate diagnosis. To understand which ocular muscle is at fault it is important to consider not so much the action of each separate muscle, as the binocular movements of the eyes, and the part which the several muscles play in relation to each other when these movements are made. The recti muscles which act in a horizontal direction have but one function, to rotate the eye about its vertical axis. When, however, the other muscles which act in vertical direction are considered, it will be seen that owing to their line of action being placed obliquely to the line of vision, each has a primary action about the horizontal axis, and subsidiary actions about the vertical and antero-posterior axes of the globe. The actions of the ocular muscles can therefore be tabulated as follows:—

	<i>Primary action</i>	<i>Secondary action</i>
Internal Rectus	Adduction	None
External Rectus	Abduction	None
Superior Rectus	Elevation	Internal rotation and intorsion
Inferior Rectus	Depression	Internal rotation and extorsion
Superior Oblique	Depression	External rotation and intorsion
Inferior Oblique	Elevation	External rotation and extorsion

The vertically acting muscles exert their maximum effect when the antero-posterior axis of the eye is in the same plane as the line of action of the muscle. Consequently, the superior and inferior recti act most strongly when the eye is rotated outwards, while the oblique muscles exert their maximum effect when the eye is turned inwards.

Krewson¹⁵ writing on the actions of the oblique muscles, said "The planes of action of the two oblique muscles were said by Maddox to be identical, each making an angle of 51 degrees with the median plane." From corrected figures of Vockmann, however, the tendon of the superior oblique makes an angle with the median plane of 55 degrees 21' while the inferior oblique muscle makes an angle of only 50 degrees 57'. This means that the inferior oblique has a greater vertical purchase on the globe than the superior oblique, *i.e.*, its plane of action is closer to the antero-posterior diameter. In fact, the inferior oblique is actually pulled more closely towards parallelism with the median plane by its check ligament, which pulls the muscle belly laterally. Thus the inferior oblique muscle contributes more of its energy to elevation than the superior oblique contributes to depression; the rate being 60-57 or 42 per cent. to 37 per cent. as indicated by Verrijp."¹⁶ These facts might account for the marked overaction of the

inferior oblique, which is often seen to occur when the superior rectus of the opposite side is paralysed, compared with the over-action of the superior oblique which follows a paralysis of the opposite inferior rectus.

Although individual muscles and their primary and subsidiary action must be described, it is necessary to emphasise that no ocular muscle acts alone. Normally they act in concert to maintain perfect ocular balance in whatever position they move the eyes.

The following are the muscles which are associated in the conjugate movements of the eyes, in the six diagnostic directions in which they exert their maximum mechanical power.

<i>Diagnostic directions</i>	<i>Muscles whose action predominate</i>	
	<i>Right eye</i>	<i>Left eye</i>
Right	External rectus	Internal rectus
Left	Internal rectus	External rectus
Upwards and right	Superior rectus	Inferior oblique
Upwards and left	Inferior oblique	Superior rectus
Downwards and right	Inferior rectus	Superior oblique
Downwards and left	Superior oblique	Inferior rectus

Each muscle of the above pair is known as the contralateral synergist of its partner, as are the two internal recti when the eyes converge. The homolateral vertical recti and obliques are direct antagonists of each other, while the superior and inferior recti are the indirect antagonists of the superior and inferior obliques respectively, of the same side.

Methods of diagnosis employed

From the point of view of surgery and orthoptics, identification of the affected muscle or muscles is essential for correct diagnosis and treatment. Such a statement may seem platitudinous, but it is a common experience to find that in many cases of diplopia the wrong muscles are incriminated.

The correct identification of the affected muscle is sometimes a matter of considerable difficulty, especially when secondary contracture of the direct antagonist of the same side has already occurred, leading to a relative paresis of the synergist of the other eye.

Speaking on the ocular palsies, Chavasse¹¹ said, "Not only are all of us free to form and express our opinions about a given case, but, and this is particularly democratic, the various opinions are allowed to have a most exhilarating diversity. If one authority says the right superior rectus is paralysed, we can be sure that another equally eminent will blame the left superior oblique." The sequence Chavasse had in mind was undoubtedly :—

1. A primary paralysis of the right superior rectus (with overaction of the left inferior oblique).
2. A secondary contracture of the right inferior rectus, leading to
3. A relative paralysis of the left superior oblique.

This sequence is often seen to follow, in a more marked degree, a primary paresis of the superior oblique and overaction of the opposite inferior rectus. With the lapse of time the inferior oblique of the same side undergoes a secondary contraction, causing a relative paresis of the opposite superior rectus. This overaction of the inferior oblique may be due in part to the anatomical arrangement of its line of action and of its check ligament, both of which peculiarities have already been mentioned. From the history of the accident it is often possible to determine into which of the three different categories the particular case will fall. The double vision which follows a frontal sinus operation will usually correctly suggest a paresis of the superior oblique of the same side.

Case 7 will illustrate the significance of the proper history. This pilot presented himself with the story that he had asked his medical officer if he could have his eyes "checked up" because he noticed that after one or two drinks he began to see double. This had never happened to him before, and he thought it would be a good thing to go along to the "Doc." He was seen by his doctor, who referred him for disposal and treatment.

The patient had a slight scar on his eyebrow and slight ptosis of the left upper lid; apart from that there was nothing unusual about his appearance. On being asked if his head had been injured recently, he said he had received a slight "bang" on the head three months ago, but thought that he had quite recovered. On taking a more complete history, it was revealed that this pilot had been shot down near the Dutch frontier, and crashing in a turnip field he had struck his head on the gunsight of his Mustang. He had treated his cut face himself, and had made what he considered to be a complete recovery. X-Ray revealed a fracture of the zygomatic malar complex with a relative paresis of the left inferior oblique and left internal rectus muscles. The history of the injury (whenever it is possible to elicit one) will, therefore, often help considerably in distinguishing to which group the case belongs.

The nature of the diplopia, whether it is horizontal or vertical in character, crossed or homonymous, will also indicate which movement of the eyes causes the greatest separation of the images, and this particular movement can be carefully watched during the next part of the investigation, which is the observation of the ocular movements.

Observations of the ocular movements

With care it is possible to make two observations during the examination of the ocular movements:—

1. The limitation of movement of the affected eye.
2. The overaction, during the same movement, of the sound eye.

It is not always possible to observe any limitation of movement in the direction of action of a paresed muscle. If, however, the sound eye is covered, the overaction of this eye, or the secondary deviation, often will be clearly demonstrable.

Chavasse¹¹ has pointed out that paralysis of an extra-ocular muscle results in overaction of the synergic muscle in the other eye. For example, an "upshoot" of the right eye which is found to occur when the eyes are directed upwards and to the left, means that the left superior rectus is partially or completely paralysed. Therefore, by this means alone, it is often possible to identify the paresed or paralysed muscle.

Since the nervous energy which is called upon to activate the paralysed muscle is also conducted to the contralateral synergist (Hering's law), the movement of the paralysed eye being less than its fellow, the image of that eye will be projected into space in the direction of action of the paralysed muscle, and farther away than the true image. Consequently, when the ocular movements are made, the diplopia will be found to be greatest in the direction of action of the paralysed or paresed muscle, and the more displaced image will be found to belong to the affected eye.

If the diplopia is horizontal in type, an object such as a pencil is moved from the midline, first to the left and then to the right; if an adductor muscle is at fault, a crossed diplopia will occur, while if an abductor is affected there will be an homonymous diplopia, more marked on the affected side.

In the case of vertical diplopia, the four positions where there is maximum action of the particular vertical muscles are examined. Namely, upwards to the right, upwards to the left, downwards to the right and downwards to the left. To identify the affected muscle it is necessary to find out where the greatest separation of images occurs. By covering and uncovering one eye it is possible to determine quickly to which eye the "farther away" image, or false image, belongs. If this image is shadowy and difficult for the patient to see, the red/green diplopia test is carried out. This is the same in essentials as that described above, except that by means of red/green goggles and a bar of light, a red image can be presented to the right eye and a green image to the left eye. The light is moved into the same six diagnostic positions, the

examiner noting where the maximum horizontal and vertical diplopia occurs.

The muscle which is affected when the diplopia is greatest in a certain position may be tabulated thus :—

1. Diplopia—horizontal.

(a) Maximum separation of image looking to the right.

Further image belonging to right eye—paralysis of right external rectus.

Further image belonging to left eye—paralysis of left internal rectus.

(b) Maximum separation of images looking to the left.

Further image belonging to left eye—paralysis of left external rectus.

Further image belonging to right eye—paralysis of right internal rectus.

2. Diplopia—vertical.

(a) Maximum separation of images upwards and to the right.

Higher image belonging to right eye—paralysis of right superior rectus.

Higher image belonging to left eye—paralysis of left inferior oblique.

(b) Maximum separation of images upwards and to the left.

Higher image belonging to left eye—paralysis of left superior rectus.

Higher image belonging to right eye—paralysis of right inferior oblique.

(c) Maximum separation of images downwards and to the right.

Lower image belonging to the right eye—paralysis of right inferior rectus.

Lower image belonging to left eye—paralysis of left superior oblique.

(d) Maximum separation of images downwards and to the left.

Lower image belonging to left eye—paralysis of left inferior rectus.

Lower image belonging to right eye—paralysis of right superior oblique.

Measurement of the diplopia

The muscle or muscles at fault having been diagnosed, it is now necessary to measure the amount of the diplopia in order—

1. to be able to observe any recovery that may be taking place;
2. to assess the results of treatment.

To do this, the following methods are employed :—

(a) The Maddox rod.

(b) The Hess screen.

(c) The synoptophore.

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(a) Maximum separation of images upwards and to the right.

Higher image belonging to right eye—paralysis of right superior rectus.

Higher image belonging to left eye—paralysis of left inferior oblique.

(b) Maximum separation of images upwards and to the left.

Higher image belonging to left eye—paralysis of left superior rectus.

Higher image belonging to right eye—paralysis of right inferior oblique.

(c) Maximum separation of images downwards and to the right.

Lower image belonging to the right eye—paralysis of right inferior rectus.

Lower image belonging to left eye—paralysis of left superior oblique.

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2. to assess the results of treatment.

To do this, the following methods are employed :—

(a) The Maddox rod.

(b) The Hess screen.

(c) The synoptophore.

The Maddox rod. It is useful, particularly during wartime, not to have to rely on elaborate equipment, and it has been found that the estimation of diplopia by the Maddox rod is simple and accurate. By means of the Maddox rod the spot light seen by one eye is presented to the other as a red line of light, and the amount of horizontal and vertical separation can be measured on the tangent scale or in prism dioptres.

The Hess screen. In essentials, this is a dissociation test, one eye seeing the red dots through a red glass, and the other the green pointer through a green glass. With the green glass before the affected eye, the pointer will be placed inside the red dots, when the eye is looking in the line of action of the paralysed muscle; the reverse will be the case when the green glass is placed before the unaffected eye. The primary deviation of the paralysed eye and the secondary deviation of the sound eye can both be charted, and the amount of recovery, either spontaneous or as a result of treatment, can be accurately assessed.

The synoptophore. Finally, the eyes of each patient suffering from diplopia are tested on the synoptophore. The angle of deviation is measured, ahead and on side movements. Slides of dissimilar objects are used to measure the "simultaneous perception angle," and slides of similar objects are used for testing the angle of fusion. The duction power of the eyes is then measured, adduction, abduction and torsion are usually recorded in degrees and height in prism dioptres. Finally, an assessment is made of the stereoscopic vision.

Treatment

When a man, after being wounded or injured in the head, becomes sufficiently aware of his surroundings to notice that he is seeing double, even although the diplopia occurs only in certain positions of the eyes, stereoscopic films of the head in the vertical position should be taken, in order to be sure that there is no bony injury of the walls of the orbit. An ordinary X-Ray picture taken in the "AP" position frequently will not show any deformation of the bones. Case No. 2 illustrates the ease with which these fractures can be missed. An X-Ray report was requested on . . . (1) the left ankle, (2) the skull. The report stated (1) fracture of internal malleolus with marked medial displacement of lower fragment. (2) No fracture seen on these films.

The surgeon later, on clinical findings, asked for stereoscopic films of the face for ? middle third fracture. The following is the X-Ray report: "Appears to be some depression of the left orbital floor, with separation of the fronto-malar suture. Left maxillary antrum is obscured presumably by blood clot." This case was

complicated by severe burns, and initially the swelling of the soft tissues closed the eyes and effectively obscured the bony landmarks.

(A) *Restoration of the normal anatomy of the parts.* Frontal blows such as pilots commonly receive when attempting a crash landing may be transmitted through the nasal arch and the ethmoids to the maxilla and the bony walls of the orbit; they result in middle third fractures of the face and are often the cause of diplopia.

The involvement of the orbit can be diagnosed before the onset of swelling, by palpation of the orbital margin which will often reveal a gap in the neighbourhood of the malar maxillary suture.

The treatment lies in the hands of the facio-maxillary unit, and consists in the reduction of the fracture and the fixing of the fragments by anchoring them to the teeth or skull. The middle third fractures of the face comprise, in the first place, the malar fracture, and secondly what Mathews¹⁷ calls the fractures of the malar maxillary complex. If seen within nine or ten days of the injury, malar dislocations can be levered back into position; if left longer, open operation and wiring of the fronto-malar processes will usually be necessary (Case 52). Fractures of the malar maxillary complex run across the outer wall and floor of the orbit to the anterior surface of the maxilla, and the malar is frequently telescoped downwards, backwards and inwards towards the maxillary antrum. The separation of the malar from the external angular process of the frontal can usually be felt, as well as the step deformity in the infra-orbital ridge, if the extensive swelling of the soft tissues has not already obliterated the bony landmarks and closed the lids.

The diplopia which results from the displacement or loss of the orbital floor may not indeed be noticed by the patient before his lids are closed by the swelling of the surrounding tissues (Case 2).

The treatment consists in raising the orbital floor by packing the antrum; this is done with 1" ribbon gauze until the pupil on the affected side is slightly higher than its fellow (Case 4).

If immediate restoration of the floor of the orbit is not effected shortly after the injury, it may be advisable in severe cases to remake the orbital floor by the insertion of a bone graft taken from the inner table of the iliac crest (Cases 4 and 5).

When the displacement has been reduced, as far as possible by these means, the residual diplopia can be dealt with by a shortening or lengthening operation on the extra-ocular muscles.

(B) *Orthoptic Treatment.* Before the surgical correction of these cases is undertaken, however, a careful investigation of the diplopia is made along the lines already described, and orthoptic

treatment is begun. The orthoptist measures each case and, if the degree of the diplopia is small and the fusion hold is good, continues with the treatment in order to increase the duction power and help the ocular rehabilitation of the patient. After operation, orthoptic exercises are again given to stabilise the binocular reflexes in the new position of the eyes.

In dealing with head injury cases, a special understanding, which is partly innate and partly bred of experience, is invaluable in treating not only the paresis of the ocular muscles, but in fanning the flames of hope and self confidence, which in many of these cases tend to burn so low (Cases 11 and 41).

A number of patients tend to make a spontaneous recovery, but in the opinion of R.A.F. ophthalmologists orthoptic exercises judiciously given cut short the period of convalescence and constitute a very valuable therapeutic measure. Many others who have made what is considered to be a good recovery, without any orthoptic treatment, are left with a residual weakness. This often causes ocular discomfort and is only partly overcome by the adoption of a head tilt. In times of nervous stress or prolonged ocular fatigue the binocular hold may be strained to the point of disruption, and diplopia results (Case 7).

(C) *Operative Measures.* Operation is not considered until five or six months have elapsed since the date of injury. Surgical adjustment of one or more of the extra-ocular muscles is then carried out, preceded and followed by orthoptic exercises. In the more pronounced cases it is usually obvious from the beginning that operative measures will have to be undertaken, and the orthoptist will quickly know if further improvement is likely to occur. In these cases it would appear that it is better to operate before a secondary contracture of the homolateral antagonist occurs and complicates the picture by producing a relative paresis of its contralateral synergist. One of the advantages of the Hess screen is the graphic way in which these secondary contractions can be charted.

Many operative procedures are possible, and may be considered successful if judged in terms of ocular comfort and the disappearance of the diplopia in the primary position of the eyes. In the case of operational pilots, however, this is not enough. A fighter pilot, who has to fly by day and night, has continually to search the sky for enemy aircraft, and must have no diplopia in any part of his visual field. It is thought, therefore, that the standards of cure in this series are high, when after treatment the particular subject has been passed fit for flying by the Central Board of the R.A.F. and satisfactory when, as in the case of non-flying men, they have returned to duty with no symptoms and no double vision in the normal position of the eyes.

Ordinarily the twelve extra-ocular muscles of the eyes work together in harmony, to maintain binocular single vision. When one or more muscles are affected the binocular machine is unbalanced and of the operations which are performed to correct the diplopia, those which equalise the balance of power in the line of action of the paralysed or paresed muscle are obviously a better mechanical solution than those which are planned to adjust it in a line directed diagonally to the line of action of the affected muscle. The recession of the contralateral synergist is, therefore, an operation which has mechanical advantages over a weakening or lengthening operation of the direct antagonist of the same side. This operation is to be preferred, however, if there has been a secondary contraction of a direct antagonist, not because it is the antagonist of the muscle primarily affected, but because it is the contralateral synergist of the muscle in the other eye which is relatively paresed. The other reason for weakening the direct antagonist is when the unopposed torsional power of the muscle is producing a marked cyclophoria.

Case 32 illustrates the degree of exocyclophoria which resulted from a relatively mild paresis of the right superior oblique. The patient complained that the tilting of the images worried him far more than the vertical displacement. The overacting inferior oblique was tenotomised with the result that the exocyclophoria was reduced by half (from 10 prism dioptres to 5) and his subjective symptoms greatly improved. Case 49 is another example of the worrying effect which the tilting of the image had upon the patient. Two operations were performed upon the eye muscles with apparent success. Two months later he complained that he began to see double again after working for two hours (he was an engine fitter). The false image appeared first of all to tilt, and then to separate. After the exocyclophoria had been surgically corrected discomfort disappeared and he became ocularly fit for aircrew duties.

In the smaller degrees of vertical diplopia, Lyle¹⁵ advocates a controlled tenotomy of the superior or inferior rectus muscle. By means of a light placed above the patient's head, and a Maddox rod held before his affected eye, the muscle can be cut in three places and allowed to stretch until the red line passes through the light. The operation has been done twice only in this series (Cases 31 and 46). On the third occasion on which it might have been performed, the muscle, in this case the inferior rectus, was recessed. There was no bleeding, and after the recession the gap between the old insertion and the new was no more than 1 to 1½ mm. This corrected 2 prism dioptres only of hypotropia (ahead) and completely abolished the troublesome diplopia which the patient

experienced on reading or walking down stairs, due to the marked overaction of the muscle. The difficulties which have been experienced with these so called controlled tenotomies seem to be due to a lack of continued control, although the immediate result may appear to be satisfactory. This it is thought is caused by:—

1. The premedication.
2. The dilatation of the pupil (cocaine).
3. The haemorrhage from the cut vessels.

In (1) the patient seems to find it difficult to localise the red line in relation to the light; (2) the wide pupil does not assist in making a nice judgment possible. (3) The haemorrhage, even though small in amount, seems to congeal and subsequently to contract; this anchors the muscle and the organising clot eventually draws it up again. This is more likely to occur if the muscle is the superior rectus, since the eyes when bandaged tend to move upwards and slightly outwards. Orthoptic exercises will in great measure counteract this tendency; a small bloodless recession, however, obviates the difficulty and should, in the author's view, be done first.

In Case No. 31 the overaction of the superior rectus was corrected by a three snip tenotomy controlled on the table by a Maddox rod, but within a week the hypertropia was a little greater than before the operation (*i.e.*, 3 P.D.). A two millimetre recession was then carried out with a satisfactory result; (*vide* Hess chart) the operation was, however, slightly more difficult and less bloodless owing to the previous reaction.

In dealing with an overaction of the inferior oblique, White in America has advocated a recession of the tendon at its attachment to the globe.¹⁹ The operations carried out in this series of cases have been of two types, (1) a tenectomy (rarely a tenotomy) of the inferior oblique near the origin of the muscle, and (2) a tenotomy of the muscle near its insertion to the globe. The good results obtained from very varying degrees of overaction of the muscle, seem in the first method due to not anchoring the muscle tendon in a fixed position. The conjunctiva is not opened, and the muscle slides along the floor of the orbit to take up a position in harmony with the other extra-ocular muscles, particularly its yoke muscle of the other side, *i.e.*, the contralateral synergist. To enable this to occur while the muscle is still in a plastic condition the eye pads are removed, the next day if possible, and the eyes balanced with fusion pictures on the synoptophore. A tenotomy near the insertion of the muscle is more drastic and seems more suitable for the higher degrees of overaction, particularly when the results of the first method have proved inadequate.

A striking example of this is afforded by Case No. 53. The

subject was a pilot who had crashed in Germany and owing to a paresis of the right superior oblique, had suffered from constant diplopia during the three years he had been a prisoner of war. During the time he spent in captivity in Stalag Luft. III he developed a certain amount of suppression of vision in the left eye, a head tilt to the right, and a secondary paresis of the right superior rectus. On his return to England he was admitted to hospital suffering from diplopia. When the eyes were relaxed there was a gross overaction of both the right inferior rectus, and the left inferior oblique (L/R 26Δ , 20° to the R.). Accordingly a generous tenectomy of the left inferior oblique and a recession of the right inferior rectus was carried out. Four weeks later there was still considerable overaction of both muscles (L/R 17Δ , 20° to the R.). The right inferior rectus was accordingly again exposed (L/R 25Δ relaxed), completely freed and tenotomised. At operation there was found to be no pull by the muscle, and the picture which presented itself was more one of generalised thickening and contracture round the muscle itself. Even after this operation there was still the overaction of the left inferior oblique to deal with, and this muscle had already been thoroughly tenectomised near its origin (L/R 8Δ , Exyclophoria 6°).

The other end of the muscle was therefore exposed and tenotomised near the globe. By this operation the exyclophoria was eliminated, and the hyperphoria reduced to zero straight ahead, and L/R 4Δ , 20° to the R.

It may be seen that either end of the inferior oblique is readily accessible, and that it sometimes is advantageous, as in this instance, to adopt the alternative method of approach.

The superior oblique muscle is not infrequently paralysed or paresed following a direct injury in the region of the pulley (Cases Nos. 15 and 38) or indirectly following a lesion of the fourth cranial nerve. The surgical treatment is a recession of the contralateral inferior rectus. Landolt⁹ advanced the inferior rectus of the same side, but this is not as satisfactory as the former operation, for the following reason: Besides the limitation of depression of the affected eye there is also some extorsion present, owing to the paresis of its secondary action which is that of intorsion. If, now, the inferior rectus of the same side is advanced, the vertical diplopia will undoubtedly be improved, but even if the obliquity of the new position is adjusted, the torsional diplopia will be aggravated, since the secondary action of the inferior rectus is that of extorsion. In regard to an overaction of the superior oblique, it must be remarked that this is a very rare condition, and its treatment by surgical means seems to be rarer still.

As a result of a crash in a twin-engine bomber the supra-orbital

margin of the left frontal bone of the air gunner described in Case 1 was fractured, and the trochlea displaced; the new position of the tendon of the superior oblique allowed the muscle grossly to overact. This caused a very troublesome diplopia when looking down, and an attempt was made to correct the overaction by lengthening the tendon of the oblique between the pulley and the globe. (Cross.) The operation did not reduce the amount of vertical diplopia, but it did reduce the incomitance so that the Hess screen showed the paresis now to be affecting more the left inferior oblique, with overaction of the right superior rectus. By means of a 3 mm. recession this overaction was satisfactorily reduced and the diplopia abolished except on high dextro-elevation of the eyes.

In connection with the surgery of the superior oblique about which little has been written in this country, it may prove of interest to recount the series of untoward incidents which befell both the writer and a patient who was admitted to hospital on account of inability to maintain single vision when using his reflector gunsight. Trauma could not be held entirely responsible for the condition, and although on a number of occasions this officer (an Irishman) had suffered from a series of minor injuries, it was thought that basically the trouble was caused by a congenital weakness of the left inferior rectus, or ? birth injury. When during his training he looked through his reflector sight in order to bring his guns to bear upon the drogue, he found it impossible to sight the target unless he closed one eye. This became increasingly noticeable if he was diving, and had to look into the sky above him: on examination he was found to have diplopia to the left, above and below eye level, particularly when looking down. There was gross overaction of the right superior oblique, paresis of the left inferior rectus and marked overaction of the left superior rectus. The first operation, namely a recession of the left superior rectus, lessened the diplopia up and to the left, but made practically no difference to the double vision experienced in the lower temporal field. Encouraged by the successful outcome of the surgical adjustment of the inferior oblique at its insertion to the globe, the improvement brought about by Cross in a similar case by splitting and lengthening the tendon of the superior oblique between the trochlea and the eyeball, and by White's description of the feasibility and practical utility of recessing the superior oblique, a lengthening operation on this muscle was undertaken. The right superior rectus was divided and reflected; the superior oblique exposed, divided and lengthened near its insertion to the globe, the superior rectus was then reattached to its insertion and the conjunctiva closed.

This operation can only be described as being disastrous in its outcome. The superior oblique apparently became adherent to the overlying superior rectus and the incyclophoria which resulted was both extraordinary and alarming. A third operation was carried out to free the adhesions, and the retaining catgut suture completely removed. Improvement resulted, but after a while, owing to the unopposed action of the superior oblique, 3 degrees of exyclophoria occurred whenever the eyes were turned to the right. The false image rose and twisted, the torsion being greater in degree and causing more discomfort than the slight vertical displacement of 2 P.D. "Clip on" prisms which enabled the patient to read in rather more than less comfort were prescribed to adjust the vertical component of the imbalance, and the patient sent on sick leave to enable the condition to stabilise sufficiently for a final adjustment to be made.

On his return to hospital the right inferior oblique was tenotomised only, and following a course of orthoptic exercises which were started the following day, the exyclophoria was eliminated.

The whole story, although it has a happy ending, had during its unfolding its chequered and dramatic moments. However, the practice evolved by Lyle and Cross in 1941, in which a recession or lengthening of the overacting synergist is employed to adjust the imbalance caused by a paresis of an extra-ocular muscle, is still upheld, and this case must be looked upon very definitely as the exception which proves the rule. (The case is described in detail in Case Records, No. 55.)

The correction of a diplopia due to a paresed vertical muscle, by attempting to strengthen it mechanically by means of a shortening operation is difficult to judge; the amount of the advancement cannot be accurately estimated owing to the variation in the amount of the paresis, and the amplitude of fusion will not compensate for a residual imbalance to the same degree as in a horizontal diplopia. Even an advancement of the synergist of the same side is not entirely satisfactory, although Stanculeanu⁸ has reported that an advancement of the right inferior rectus, in cases where the right superior oblique has been paralysed, has given satisfactory results. In those cases of paresis of the lateral recti, a recession of the overacting muscle of the opposite side seems to balance the extra-ocular muscles better, and to interfere less with the mobility of the eyes than operations designed to increase the leverage effect of the paresed muscle.

Case 44 is an example of a paresis of the left external rectus which was still present 18 months after a bomb injury. There was no binocular vision when examined on the Bishop Harman diaphragm test, and a lateral excursion of 15 degrees only in the

left eye; binocularly there was marked overaction of the right internal rectus when the patient was looking to the left, with immediate diplopia. The right internal rectus was recessed, and the patient was discharged, holding his head straight, maintaining binocular single vision, and suffering from no symptoms except on extreme laevo-version. This seemed to be a very satisfactory result, until 7 months later when the patient was readmitted to hospital suffering from headaches and a recurrence of the diplopia. The left internal rectus was recessed and subsequently the leverage of the right internal rectus was again reduced. It is interesting to note that the power of adduction compared with the previous figure of 25 degrees, was not impaired by these proceedings, and that orthoptic treatment to reinforce the binocular reflexes was given before and after each surgical operation. On discharge from hospital this airman's symptoms were completely relieved and, some 18 months later, had not returned.

In dealing with a complete paralysis of the external rectus, it is possible not only to straighten the eye, but to obtain a fair range of lateral movement by the method originated by Hummelsheim, but more often associated with the name of O'Connor. On general principles Cross considered that recovery from the injury might, in the early months, be aided by reducing the drag on the paralysed muscle by means of a recession of the homolateral antagonist. This contention, although undoubtedly sound in theory, could not in practice be substantiated, as in the three cases so treated in hospital the external rectus remained completely paralysed. For the result illustrated in Case No. 55, an advancement and resection of the paralysed muscle was carried out, and equal strips were taken from the vertically acting recti and attached to its insertion; in addition to the internal rectus of the same side, which had been recessed some five months previously, the internal rectus of the opposite side was similarly dealt with and the ultimate range of external movement increased to 15 degrees. It seems important to take strips of equal width and length from the muscles above and below. In one case where unequal strips were taken to overcome a vertical imbalance which was also present, a troublesome hypertropia developed when the eyes were turned to the affected side. Subsequent operations for the correction of this hypertropia resulted in the loss of the extra lateral movement which had previously been obtained but had been associated with a vertical tropia.

A muscle may be looked upon as a lever of the third degree with power acting between the fixed point, or fulcrum, and the weight to be moved. From the above description it may be seen

that the surgical measures that have proved most efficacious may, in their order of priority, be listed as follows:—

1. Reduce the leverage of

- (i) the synergist of the opposite side;
- (ii) the direct antagonist of the same side;
- (iii) the indirect antagonist of the same side;
- (iv) the corresponding muscle of the opposite side.

The required effect may be produced by:—

- (a) A recession of the muscle tendon.
- (b) A tenotomy or tenectomy.
- (c) A three snip extension of the tendon.

2. Increase the leverage of

- (i) the paresed muscle;
- (ii) the synergist of the same side.

The required effect may be produced by:—

- (a) An advancement of the muscle.
- (b) An advancement and resection.

Since the advanced muscle will undoubtedly adhere to the globe at the site of its original insertion, from a mechanical point of view it will have the same effect as a resection.

Particular care must be taken to avoid over-correction, but if this occurs the same procedure is adopted; the primarily affected muscle now taking the place of the contralateral synergist which, on account of the mechanical interference, may be considered to be suffering from a relative paresis.

The corresponding operations are therefore:—

	For under-correction	For over-correction
The reduction of the leverage.	The contralateral synergist.	The paresed muscle.
The reduction of the leverage.	The direct antagonist of the same side.	The indirect antagonist of the opposite side.
The reduction of the leverage.	The corresponding muscle of opposite eye.	The synergist of the same side.
The increase in the leverage.	The synergist of the same side.	The corresponding muscle of the opposite side.

Here, it may be advocated that when, owing to secondary contracture, there is an overaction of the inferior oblique as the direct antagonist of the same side, as well as the opposite inferior rectus as the contralateral synergist, the inferior oblique should be dealt with first, and the final adjustment carried out on the vertical rectus. For example, following a paresis of the right superior oblique, a contracture of the direct antagonist, the right inferior oblique may have taken place. Before the operation of the contralateral synergist (the left inferior rectus) is dealt with, it is

advisable to perform a tenectomy of the right inferior oblique, and to adjust the residual imbalance by a recession or a three-snip tenotomy of the left inferior rectus at a later date.

Finally, it may perhaps be emphasised that in the writer's experience gentleness and the arrest of haemorrhage are two factors which, without putting too high a tax on time, definitely repay the surgeon and hasten convalescence. By preventing blood clot and adhesions forming at the site of the operation the mobility and surgical adjustment of the eye is unlikely to be upset by subsequent contraction.

As in a game one plays for a leave, the surgeon in these matters endeavours to be well placed for the winning stroke, lest perchance the accuracy of his former efforts has not achieved finality.

The different types of injury, the methods of treatment and the results obtained are given in Tables I and II.

Twelve typical case histories of traumatic diplopia have been selected and one congenital case included as affording a good example of the outcome of surgical adjustment of an overacting superior oblique muscle. These form an appendix to this paper.

Analysis of cases

The cases have been divided into three main categories, according to the part affected by the injury. Some of the cases fall naturally into their appropriate classes; others, in which the damage has been more widespread, properly belong to more than one group. A consideration of the history, the course and the final disposal has, however, usually been sufficient to determine to which group the case belongs. To take a concomitant concussion as an indication of nerve injury is often misleading and inconclusive. Aldren Turner²⁰ writing on the indirect injuries of the optic nerve says "considerable damage can be done to the intracranial structures without the general cerebral disturbance which results in concussion, a fact equally true of the brain itself where extensive damage can be inflicted without loss of consciousness, when the impact is from an object of small size, such as a bomb fragment or a piece of masonry."

Case 37 illustrates, somewhat dramatically, the transitory nature of a concussion, which was followed by a lesion of the mid-brain. The airman described was flung out of an aeroplane, striking his head as he fell against some part of the aircraft. This initiated a lesion in the mid-brain which later caused diplopia and a transient paralysis of the legs. He could not have been unconscious for many seconds, since he effectively pulled the rip cord of his parachute and made a satisfactory landing. On the other hand, the patient described in Case 4 was unconscious for 12

hours, although subsequent examination showed that the left orbital floor, and not the intracranial contents, had suffered the greater damage.

From an analysis of these cases it appears that a post traumatic diplopia is more often the result of intra-orbital than of intracranial injury; with this view Cross²¹ is in agreement. Many cases that have made a "spontaneous" recovery have a residual weakness of the previously affected muscle. Others are left with a troublesome, and often in the case of aircrew, a disabling diplopia, which is more marked in certain positions of the eyes.

In three cases of the series, immediate treatment was given to restore the normal anatomy of the parts. In Case 4 the antrum was packed in order to maintain the elevation of the floor of the orbit.

In Cases 3 and 52 the malar was wired. See Table II. Patients with head and maybe other injuries are usually attended first by a general surgeon who is often more exercised by considerations affecting concussion and general injuries, than by thoughts of diplopia and its possible treatment. Only three patients, therefore, in group I received immediate treatment for the fractures involving the orbits, and many of the cases had, it appeared, been overlooked altogether (e.g., Case 6, Table II). To do this as has been explained already, is very easy, unless the possibility of a mid-face fracture is borne in mind and stereoscopic X-Ray pictures, in the vertico-mental position, are taken.

As regards the late treatment of these cases, two patients (Cases 4 and 5, Table II) received at the plastic centre a bone inlay from the crest of the ilium, to raise the floor of the orbit (Photo. Case No. 5), and the others, except in cases of refusal, or gross damage to the central nervous system, were satisfactorily treated by orthoptic exercises and surgical adjustments of the extra-ocular muscles.

The attached Table I shows the muscle or muscles that were affected, and those that were adjusted in order that diplopia might be eliminated over the widest range of ocular movement. The Maddox rod readings at 6 metres, before and after operation, have been given with the subject looking straight ahead. These would appear to be more graphic and concise than the synoptophore readings which are included in the records of each case.

Eleven patients were treated with orthoptic exercises alone; of this number six were pilots and one was a navigator. The six pilots all returned to flying, but the navigator, who would not consent to an operation, could not be sent back to flying on account of a residual imbalance. The six pilots, in spite of making a good "spontaneous" recovery, had a residual weakness which

made it difficult for them to continue with their flying duties. The fact that orthoptic exercises cured their symptoms and enabled them to return to flying, cannot be denied. For that reason such exercises, when given by an orthoptist with experience of the service, must be considered a valuable form of treatment, and one that can be relied upon, not only to re-educate the ocular muscles, but to improve the whole outlook of the patient.

In all the other cases the synoptophore has been used before and after operation, both to check the measurements of the case, and to strengthen by carefully graduated exercises the binocular reflexes.

Of the 50 extra-ocular muscles which were adjusted, 28 operations were performed upon the contralateral synergist or agonist, and 14 upon the direct antagonist of the same eye. On six occasions the corresponding muscle of the opposite side was adjusted, and other operations numbered two; in one of these the paresed muscle itself was advanced; this procedure was found to be unsatisfactory and on that account was not repeated, except in the O'Connor's operation, where the affected muscle was completely paralysed. (See photograph, Case No. 54.)

The incidence of single and double muscle paresis is shown in this series of 54 cases, in the tables A and B.

TABLE A.

Paresis of a single muscle.

	Superior rectus	Superior oblique	Inferior rectus	External rectus
Right	4	14	2	1
Left	10	3	0	1

TABLE B

Paresis of two muscles.

	Elevators	Depressors	External recti	Internal recti
Right	4	1	2	2
Left	4	1		

It may be seen that, taking the numbers together, the muscles most often affected are those which lie under the roof of the orbit, and act in a vertical direction. White and Brown²² writing in the Archives of Ophthalmology on the occurrence of vertical anomalies, say that in a study of 11,600 persons, 715 had a vertical anomaly, and of the single muscles, the superior rectus was the muscle most affected. It is not surprising therefore, that in cases of injury to the head, the superior rectus should be the muscle which is found to be the one which is most often paresed. The fact that the inner edge of the superior rectus separates itself from the rest of the muscle relatively late in development, to form the levator palpebrae superioris, may

account for the constitutional weakness of the muscle, which shows itself as a paresis when the other muscles, supplied by the third nerve, escape.¹² Cross²¹ in a survey of the causes of diplopia at a Military Hospital for head injuries, found that in 138 cases the extra-ocular muscles were affected in the following order: superior rectus, 24 per cent.; superior oblique, 21·5 per cent.; external rectus, 11 per cent.; inferior oblique, 6 per cent.; internal rectus, 5 per cent. and inferior rectus, 2 per cent. Various combinations of muscles accounted for the remaining percentage.

It may be seen therefore, from an analysis of these injuries, that of the cases that eventually need surgical measures for their correction, by far the greater number will need to have adjustments made to the vertically rather than to the horizontally acting muscles.

Results

One marine and nine members of the army were treated by the methods described; of these, seven returned to duty and two were invalided from the service, one for neuropsychiatric reasons, and the other for persistent diplopia for which he would have no operative treatment. Of the remaining 44 cases, 18 belonged to the ground staff of the Royal Air Force, and 26 were flying men. Of the ground staff, all returned to work with binocular vision, and no ocular symptoms. There was one exception, a girl engaged on precision work in the Women's Auxiliary Air Force. This girl had met with a severe head injury, which had resulted in a paralysis of the third and seventh cranial nerves. Although after operation she had no diplopia when looking straight ahead, and pluckily returned to duty for five months, she found the work too much for her, and was invalided from the service.

Of the 26 flying men, two later underwent hospital treatment for other injuries, two were treated at rehabilitation centres, one was invalided from the service on account of his head injuries, and one, an ex prisoner-of-war, was released from the Royal Air Force after his diplopia had been successfully treated. Two others were repatriated to the Dominions, and three regraded in a non-flying capacity on account of their nervous condition (both organic and functional) following their injuries. The remaining 15 returned to flying duties; these comprised four members of air crew, and 11 pilots.

Of the total number 73 per cent. returned to duty and 57·6 per cent. of the aircrew are known to have resumed flying. From an ocular point of view, several others are known to be fit for flying, but owing to various causes, such as being repatriated to their own country, it has not been possible to determine their

ultimate disposal. Although the estimate is a conservative one, it is thought that these figures show a distinct advance over the "expectant" method of treatment, with its two alternatives of prisms or a frosted glass.

Conclusions

This dissertation is, in its essentials, an apologia for the active and more radical treatment of the diplopia, which has been shown to be a not infrequent result of an injury to the head or face.

Owing to conditions of war, the incidence of diplopia following such injuries has risen steeply, and whereas before the war there was no recognised technique for the treatment of these cases, such a technique has now been worked out and is described in this thesis.

Similar cases collected before the war indicate that the condition will, with the advent of peace, continue to be a problem, when the complicated mechanisms of modern times again become responsible for the safety of so many of our journeys.

The methods of diagnosis and treatment which are advocated have now been well tested, and a medical board has in the case of pilots and air crews, assessed the results in relationships to the high standards of muscle balance which are required in the Royal Air Force.

When dealing with the vertically acting muscles it is, however, not necessary to obtain orthophoria in order to relieve effectively the most nauseating and distressing of symptoms, namely those of constant diplopia. Once the vertical deviation has been reduced by operation to manageable proportions, the binocular reflexes, strengthened by orthoptic training, will enable fusion to be maintained within the range of normal ocular movement. If the deviation is considerable the correction should be carried out in stages, rather than risk over-correction and another relative paresis which may well prove more troublesome both to the patient and to the operator than the first. By the aid of the Hess screen and synoptophore readings, the progress made with orthoptic exercises while the muscles are still in a malleable condition, can be watched and a second operation can, if necessary, be planned to correct any residual defect.

Experience has shown that it is rare to find a complete paralysis of a muscle following a head injury; usually it is a muscular paresis which causes the diplopia, and this more often follows a "closed" injury than a penetrating head wound.

In addition to the blurred vision and the mental confusion which the diplopia produces, there is usually an associated headache from the constant strain of trying, in some position of the

head and eyes, to fuse the images and obtain the relief of single vision. To be told that nothing can be done for him tends to deepen a patient's depression (vide Case 2) and cause neurotic symptoms to be superimposed upon his physical state. It is in these cases that so much can be effected by the treatment towards the mental as well as the physical rehabilitation of the patient. If operation has eventually to be performed, orthoptic exercises will prevent suppression in the affected eye and maintain during the waiting period the morale of the patient, as well as the tone of the extra-ocular muscles. After operation the exercises will strengthen the binocular reflexes and increase, while the eyes are still in a plastic condition, the fusion hold of the newly restored binocular vision.

The successful results of the treatment described are shown in the case records themselves in the analysis of the diplopia and in the methods used to overcome it.

The object of the work is to show that such treatment is reasonable in theory and sound in practice, and it is hoped that with a wider recognition of the possibilities of treatment, others may find relief and be restored again to a full life and binocular single vision.

Summary

1. Traumatic diplopia is not an uncommon sequel to a head injury.
2. Diplopia is caused usually by a paresis and not a paralysis of an extra-ocular muscle.
3. In the majority of cases, the "closed" head injury, and not the penetrating head wound, is the cause of diplopia.
4. The vertically acting muscles are most often affected.
5. Injuries to the orbit tend to be overlooked owing to the severity of other injuries, or masked by the swelling of the surrounding tissues.
6. The X-Ray diagnosis of fractures of the floor of the orbit is difficult to make, unless stereoscopic pictures are taken in the vertico-mental position of the head.
7. In fractures involving the orbit, the restoration of the normal anatomy of the parts is of immediate importance.
8. The orbital floor can be built up by means of a bone inlay, if immediate restoration has not been effected.
9. Well planned eye muscle surgery, aided by orthoptic exercises, is the best means of overcoming the residual diplopia and of restoring binocular single vision.

I should like to acknowledge my indebtedness to my ophthalmic colleagues, particularly to A. G. Cross and G. W. T. Cashell,

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TABLES AND CASE NOTES

In the subsequent tables and case notes the following abbreviations have been employed.

Muscles.

R.S.R. and L.S.R.	Right and left superior rectus
R.E.R. and L.E.R.	Right and left external rectus
R. Int. R. and L. Int. R.	Right and left internal rectus
R. Inf. R. and L. Inf. R.	Right and left inferior rectus
R.S.O. and L. S. O.	Right and left superior oblique
R. Inf. O. and L. Inf. O.	Right and left inferior oblique
R/L and L/R	indicate	right and left	hyperphoria respectively

Maddox rod measurements are given in prism dioptres.

Synoptophore measurements:—

Lateral deviation in degrees.

Vertical deviation in prism dioptres.

F.R. and F.L.=fixing right and left eye, respectively.

Cyclophoria is measured in degrees.

TABLE I. *The treatment of traumatic diplopia by surgical adjustment of extra-ocular muscles, showing the Maddox rod readings at 6 metres before and after operation.*

Case No. Duty Age	Muscles affected and cause	Muscles adjusted	Maddox rod		Remarks
			Before	After	
1 W/Op Air Gun 22	R. Inf. R. & R.S.O. Aircraft crash	1. L.S.O. (2) R.S.R.	R/L 8Δ-10Δ	R/L 0Δ-3Δ	Residual diplopia only on extreme dextro-elevation. For further plastic repair of face.
2 Pilot 21	L.S.R. & L.E.R. Aircraft crash	R. Inf. O.	R/L 10Δ Eso. 4Δ	R/L 2Δ	Fracture of orbital floor, revealed only by stereo X-ray. For further plastic repair of face.
8 Pilot 27	L.S.R. Aircraft crash	R. Inf. O.	R/L 4Δ	R/L 2Δ	Diplopia on laevo-elevation before operation. None after. Resumed flying.
10 Arm- ourer 35	R. Inf. R. Contracture of right superior rectus, follow- ing operation for detached retina.	(1) R.S.R. (2) L. Inf. R.	R/L 3Δ-12Δ Eso. 9Δ	Ortho.	Diplopia only on extreme dextro-elevation, and depression after operation. Return to duty.
12 Gun- ner 22	R.S.O. Direct injury to eye, with de- tachment of retina.	L. Inf. R.	R/L 3Δ-4Δ	Ortho.	Residual diplopia only on extreme laevo depression. Returned to duty.
13 Ob- server	R.S.R. Direct eye in- jury with lacer- ation of left lower lid and adhesion round L.I.O.	L. Inf. O.	L/R 2Δ	L/R 1Δ	Cyclophoria and diplopia on dextro-elevation before operation. None after. Returned to flying duty.
14 Air- crafts- man 33	R. Inf. R. & R.S.O. Piece of wood penetrated right orbit.	R.S.R. & R. Inf. O.	R/L 15Δ	R/L ½-3Δ	No diplopia with glasses after operation. Prisms worn previously. Returned to duty.

TABLE I—continued

Case No. Duty Age	Muscles affected and cause	Muscles adjusted	Maddox Rod		Remarks
			Before	After	
15 Pilot 22	R.S.O. Aircraft crash. Injury of frontal bone over right trochlea.	(1) Trochlea freed. (No benefit.) (2) L. Inf. R.	R/L 4Δ-6Δ	R/L 2Δ-3Δ	Diplopia caused by deep scarring over trochlea, from crash injury. Dip- lopia cured by second operation. Repatriated to Australia.
16 Fitter Eng. 32	L.S.R. Result of an ob- literated operation on right frontal sinus two years ago.	R. Inf. O.	R/L 8Δ Exo. 3Δ	R/L 2Δ Eso. 2Δ	Diplopia caused by oper- ation on Rt. frontal sinus. Diplopia cured by Op. or R. Inf. O. Returned to duty.
18 Pilot under- train 22	R.S.O. Aircraft crash. Compound frac- ture of the right frontal bone.	R. Inf. O.	R/L 12Δ-15Δ	R/L 4Δ	Diplopia after operation for cerebral abscess following compound frac- ture of frontal bone. Diplopia cured by R. Inf. O. adjustment. To ground duty.
19 Pilot 23	L.S.R. Air accident.	R. Inf. O.	R/L 1Δ Eso. 3Δ	Ortho.	Diplopia on laevo-eleva- tion before operation. None after. Returned to flying.
20 Ser- vice Police 25	R.S.O. Bullet wound.	L. Inf. R.	R/L 3Δ-4Δ	R/L 1Δ	Shot through right eth- moid and frontal sinus, resulting in diplopia on laevo-version. Symptom- less after operation. Returned to duty.
24 Mar ine	R.S.O. Battle injury.	R. Inf. O.	R/L 14Δ-18Δ	R/L 4Δ	No diplopia. Returned to duty.
25 Sol- dier	R.S.R. Boxing Match.	L. Inf. R.	R/L 16Δ	R/L 2Δ-4Δ	Previously told nothing could be done. Now very pleased with eyes. Returned to duty.
29 Sol- dier	R. Inf. O. & R.S.R. Bicycle accident	L. Inf. O.	L/R 2Δ	L/R 1Δ	Diplopia on elevation before operation. No ocular symptoms after. Returned to duty.
30 Flight Eng. 23	Bilateral paresis external recti. Aircraft crash.	(1) R. Int. R. (2) L. Inf. O.	Eso. > 30Δ L/R 5Δ	Eso. 8Δ L/R 2Δ	Result of operation good. Resumed flying.

TABLE I—continued

Case No. Duty Age	Muscles affected and cause	Muscles adjusted	Maddox Rod		Remarks
			Before	After	
31 Pilot 22	L. Inf. O. & L.S.R. Aircraft crash.	(1) R.S.R. 3 snip (2) R.S.R. Re- cession 2 mm.	R/L 2Δ-3Δ	Ortho.	Diplopia on dextro-elevation before operation. Result of operation good. Resumed flying.
32 Air- crafts- man 38	R.S.O. Bicycle accident Fracture of skull	R. Inf. O. for the excyclo- phoria, rather than the hyper- phoria.	R/L 1Δ Excyclo. 9°	R/L 1Δ Excyclo. 4°	Improved after operation. For encephalography.
33 Army Officer	R.S.O. Car accident.	R. Inf. O.	R L 12Δ Eso. 6Δ	R/L 3Δ Eso. 2Δ	Residual diplopia only on extreme laevo-depression. No further operation desired. Invalided.
34 Inst. Rep. 29	L.S.O. Bicycle accident.	(1) R. Inf. R. (2) L. Inf. O. (3) R. Inf. R.	L/R 12Δ-14Δ Exo. 3Δ Excyclo. 10°	R/L 1Δ-3Δ Eso. 4Δ No excyc.	Returned to work.
35 Tele- printer 19	3rd nerve palsy of right eleva- tors. Bicycle accident, cranio- cerebral injury	L.S.R. & L. Inf. O.	Eso. 9Δ L/R 5Δ	Eso. 5Δ	Diplopia persisted L/R on depression. R/L on elevation. Invalided.
39 Sold- ier 36	L.S.R. Knocked out in a fight.	R. Inf. O.	R/L 18Δ Eso. 6Δ	R/L 2Δ Eso. 4Δ	Residual diplopia only on extreme laevo-elevation. No symptoms. Return to duty.
40 Bal- loon Op. 24	L.S.R. Air raid casualty.	R. Inf. O.	R/L 3½Δ	R/L ½Δ	To rehabilitation unit.
41 Pilot 22	L.S.R. Air crash. Unconscious 4 days.	R. Inf. O.	R/L 2Δ-5Δ Eso. 2Δ Excyclo. 4°-7°	L/R 1Δ Eso. 2Δ No cyclo.	Satisfactory results. Resumed flying.
44 Flight Mech. 34	L. Ext. R. Knocked against wall by 500 lb. bomb.	(1) R. Int. R. (2) L. Int. R. (3) R. Int. R.	Eso.— 27Δ-30Δ R/L 1Δ	Eso.— 11Δ-14Δ	Operation for diplopia on laevo - version. Binoc. range increased to 15° R. to L. Returned to duty.
46 Bom- badier	Bilateral Ext.R. Bilateral Sup.O Motor cycle accident.	L. Inf. R.	Eso. 6Δ R/L 1Δ-2Δ	Ortho.	Operation for diplopia on depression, particularly to left; Residual diplopia only on extreme depression after operation. To duty.

TABLE I—continued

Case No. Duty Age	Muscles affected and cause	Muscles adjusted	Maddox Rod		Remarks
			Before	After	
47 Pilot 30	R.S.R. Air crash. Fracture of both malar bones.	L.S.R.	Eso. 6Δ L/R 1½Δ	Eso. 3Δ L/R ½Δ	Diplopia on version following a/c crash re- sulting in mid third frac- ture of face. Improved after operation. Repatriated.
48 Soldier	R.S.O. Battle casualty.	(1) L. Inf. R. (2) R. Inf. O.	R/L 6Δ-9Δ Eso. 3Δ	Ortho.	Returned to Unit.
49 Flight Eng. u/t. 24	L.S.R. Head injury at ice hockey.	(1) R. Inf. O. (2) L. Inf. R. (3) L. Inf. O.	R/L 8Δ	Ortho.	Ocularly fit flying after operation, but unfit on account of head injuries.
50 Pilot 34	L. Sup. R. Air crash.	R. Inf. O.	Excyclo. 4° R/L 1Δ	Eso. 3Δ No cyclo.	Operation for cyclo- phoria. Resumed flying after operation.
52 Pilot 22	L.S.R. & L. Inf. O. Aircraft crash. Fracture of left maxilla.	R.S.R. & R. Inf. O.	R/L 10Δ-16Δ In cyclo. 10°	Ortho.	Operation for cyclophoria on dextro-version and diplopia above eye level. Resumed flying after operation.
53 Pilot 23	L.S.O. Aircraft crash.	R. Inf. R. & L. Inf. O.	L/R 35Δ Eso. 7Δ Excyc. 6°	L/R 3Δ-5Δ Eso. 5Δ	Residual diplopia on dextro-depression only. Discharged P.O.W.
54 Soldier	R.E.R. Complete par- alysis. Battle casualty.	(1) R. Int. R. (2) L. Int. R. (3) "O'Connor's" muscle trans- plantation from right superior & inferior recti.	No measurements possible. approx. +40°	Angle 40°	Movements increased to 15° to left. Eso. 5Δ ahead. No diplopia. Invalided.

TABLE II—10 Examples of Group I (Diplopia caused by derangement of the normal bony architecture around the eye)

TREATMENT OF TRAUMATIC DIPLOPIA

613

No.	Age	Occupation	Injury	Diagnosis	Treatment	Synoptophore angle. At centre, and to side where greatest deviation occurred		Maddox rod		Result and Disposal
						Before treatment	After	Before	After	
1*	22	Air Gunner	Aircraft crash. Compound fracture L. frontal bone	Displacement of trochlea of L. Sup. oblique with overaction of L. depressors	(1) Lengthening of L.S.O. (2) Recession of R. Sup. rectus	Centre 0° R/L 5Δ—7Δ to Right R/L 7Δ—8Δ	0° R/L 4Δ 0° R/L 4Δ	Exo. 1Δ R/L 8Δ—10Δ	FR. R/L. 0Δ—1Δ FL R/L. 2Δ—3Δ	No diplopia with all normal eye movements. For further plastic operation.
2*	21	Pilot	Aircraft crash. Burns-fracture of both malar bones and floor of L. orbit	Obstruction of L. naso-lacrimal duct and paresis of L. sup. rectus	(1) Dacryocysto-rhinostomy (2) Tenectomy of rt. inf. oblique	Centre + 1° R/L 7Δ—8Δ to Left 3° R/L 16Δ—18Δ Exeyclo. 4Δ	0° R/L 1Δ	R/L 10Δ	do.	
3	24	Pilot	Aircraft crash. Facial injuries	Fracture of L. malar, Paresis of L. sup. rect. L. sup. oblique	(1) Elevation of L. malar (2) Orthoptic exercises	No immediate measurements possible	At centre and to sides-orthophoric	— to L/R 1Δ	No diplopia. Resumed flying. At last check had flown 100 hrs. since discharge.	
4	26	Turner	Knocked out in a fight	Fracture of L. maxilla. Compression of floor of orbit. Limitation of elevation. L.E.	(1) Elevation of zygoma (2) Packing of L. antrum (3) Bone platform to floor of L. orbit	No immediate measurements possible	Centre + 1° R/L 1Δ—2Δ Fixing R. & L. to Left R/L 3Δ—4Δ Fixing R. & L.	L/R 1Δ—2Δ Fixing R. & L.	Returned to his profession work—orthophoric ahead but some vertical diplopia up and down.	
5	26	Wireless Op.	Machine gun bullet through nose to L. temple	Paralysis of L. depressors. Fractured nasal and lacrimal bones and floor of L. orbit ruptured. Choroid.	(1) Dacryocysto-rhinostomy (2) Bone graft and chips to raise floor of L. orbit	Left eye 7 mm. below right	Some over-correction	No binocular vision	Cosmetic result—the retinal damage and hypertropia caused suppression of L. vision.	

TABLE II—continued

No.	Age	Occupation	Injury	Diagnosis	Treatment	Synoptophore angle. At centre, and to side where greatest deviation occurred		Maddox rod		Result and Disposal
						Before treatment	After	Before	After	
6	26	Aircraft man	Struck on face by 250 lb. bomb	Unreduced fracture of L. malar bone, paresis of L. Inf. R.						Fracture of L malar bone was missed. When admitted to hospital, diplopia not very troublesome owing to partial atrophy of L. optic N.
7	30	Pilot	Shot down, 3 months ago crash landed. Hit face on gunsight	Old fracture of L. malar. Defective elevation of left eye	Orthoptic exercises	Centre 0° R/L 4Δ To L. & R. — 1° R/L 4Δ Adduction + 10° only	Same but adduction power increased to + 35°	R/L 1Δ—fixing R. R/L 2Δ—fixing L.	same as before	Much improved but not cured by orthoptic exercises. Can now read for 3 hour. Returned to non-operational flying.
8	27	Pilot	Aircraft crash in desert	Fracture of R. frontal bone. L. maxilla and nasal bones. Paresis of L. sup. rectus.	Tenectomy of right inferior oblique	Centre 0° R/L 6Δ to Left 0° R/L 10Δ	0° R/L 2Δ—3Δ in all positions	R/L 6Δ—7Δ Exo. 2Δ	R/L 2Δ—3Δ Exo. 1Δ	No diplopia. Resumed flying (non-operational).
18*	22	Pilot u/t	Aircraft crash	Compound fracture of R. frontal bone	Tenectomy of right inferior oblique			R/L 12Δ—15Δ	R/L 4Δ	Not a fully trained pilot, returned to ground duties. No diplopia.
52*	22	Pilot	Aircraft crash	Fracture of left maxilla with paresis of L. elevators	(1) Wiring of front. to malar suture (2) Adjustment of right elevators	Centre R/L 9Δ—11Δ to Right —5° R/L 15Δ	Centre R/L 1° 5Δ to Right —5° R/L 5Δ	R/L 10Δ—16Δ —F.R. R/L 5Δ—F.L.	Orthophoric	No diplopia. Resumed flying (non-operation).

* Also included in Table I under muscle adjustments.

HISTORY.

PRE-OPERATIVE EXAMINATION. January 1, 1945.

Vision :—R. 6/5. L. 6/5.

Movements :—Defective depression of the right eye and gross overaction of the left.

Diplopia :—Vertical, with maximum separation of images on dextro-depression, lower image belonging to the right eye.

Maddox rod :—Fixing right—R/L 8 pd. Exophoria 1 pd.

Fixing left—R/L 10 pd. Exophoria 1 pd.

Synoptophore :—S.P. angle—fixing right—centre ... 0° R/L 4-6 pd.

to right ... R/L 7-8 pd.

to left ... R/L 4-6 pd.

fixing left—centre ... 0° R/L 5–7 pd.

to right ... R/L 7-8 pd.

to left ... R/L 5-7 pd.

Left incyclophoria 3°

Fusion 0° . R/L 4Δ : can infraduct to 0

Adduction to $\pm 30^\circ$

Abduction to -2°

Hess chart 1.—Right eye—paresis of right inferior rectus and superior oblique.

Left eye—overaction of depressor muscles.

Diagnosis :—Displacement of trochlea of left superior oblique.

OPERATION. January 26, 1945.

Slide lengthening of tendon of left superior oblique between trochlea and the globe.

POST-OPERATIVE MEASUREMENTS. February 1, 1945.

Maddox rod :—R/L 8 pd. Exophoria 2 pd. Fixing right and left.

Diplopia :—Now corresponds, mainly, to paresis of left inferior oblique; slightly, to paresis of right inferior rectus. Hess chart 2.

OPERATION. February 7, 1945.

Recession of right superior rectus 3 mm.

POST-OPERATIVE MEASUREMENTS. February 16, 1945.

Maddox rod :—Fixing right ... R/L 0Λ-1Λ.

Fixing left ... R/L 2 Δ -3 Δ .

Maddox wing :—Exophoria 2°–4°. No hyperphoria.

Diplopia :—Only on looking well up to the right.

Synoptophore :—S.P. angle—centre ... 0° R/L $\frac{1}{2}$ pd.

to right ... 3° R/L- $\frac{1}{2}$ pd.

to left ... 0° R/L $\frac{1}{2}$ pd.

Fusion — 0°. No hyperphoria.

Adduction $\approx 50^\circ$

Abduction to -5° .

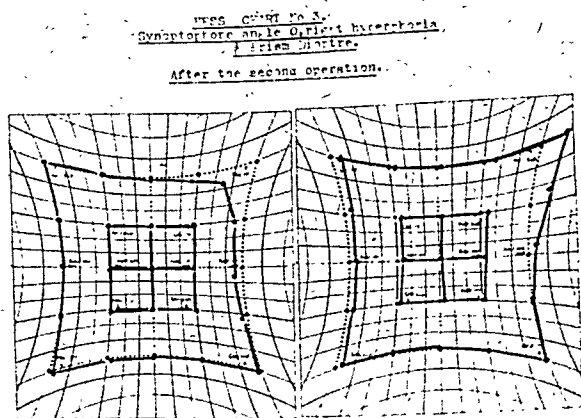
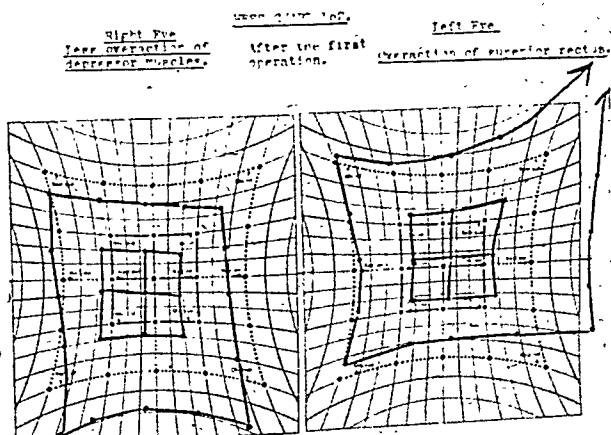
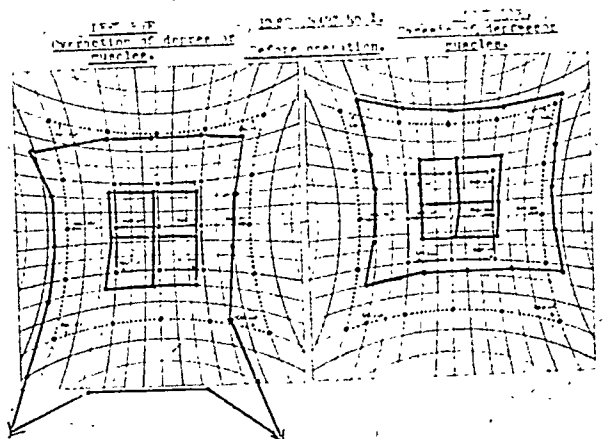
Stereoscopic vision—full.

Hess chart 3 :—Slight deviation on extreme dextro-elevation.

DISPOSAL.

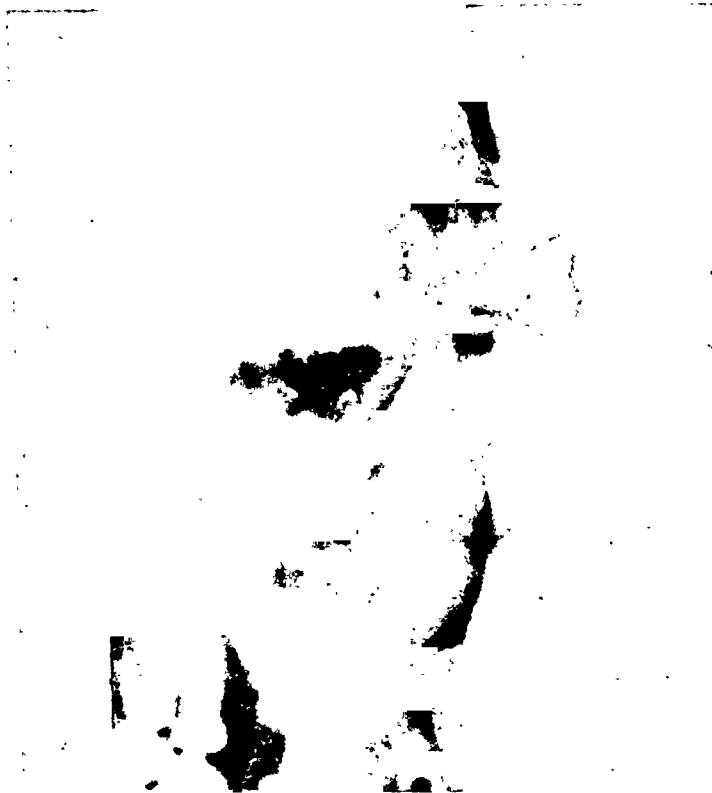
To return to the plastic centre after convalescence, for further treatment. No ocular symptoms.

CASE 1



CASE 1

Compound fracture of the left frontal bone; forward dislocation of the lower fragment.



Showing the supra-orbital margin, after it had been re-shaped by a plastic operation, and the displaced position of the left eye, before the operations on the extra-ocular muscles.

Case No. 2.



No. 1. X-RAY PHOTOGRAPH IN THE "FRONTAL" POSITION.
CONTACT TO SINUS.



No. 2. X-RAY PHOTOGRAPH IN THE "FRONTAL" POSITION.
Distortion of left orbital floor. Dislocation of fronto-orbital
suture. Maxillary antrum is obscured, presumably by fluid clot.



No. 3. X-RAY PHOTOGRAPH IN THE "FRONTAL" POSITION.
CONTACT TO SINUS.

CASE 5.

FRACTURE OF LEFT MALAR AND MAXILLA.



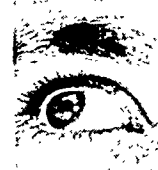
FLOOR OF THE LEFT ORBIT 1 1/2 IN. BELOW BRIDGE OF NOSE
FROM THE BRIDGE OF NOSE

Before the bone inlay

On leaving hospital.



CASE 13.

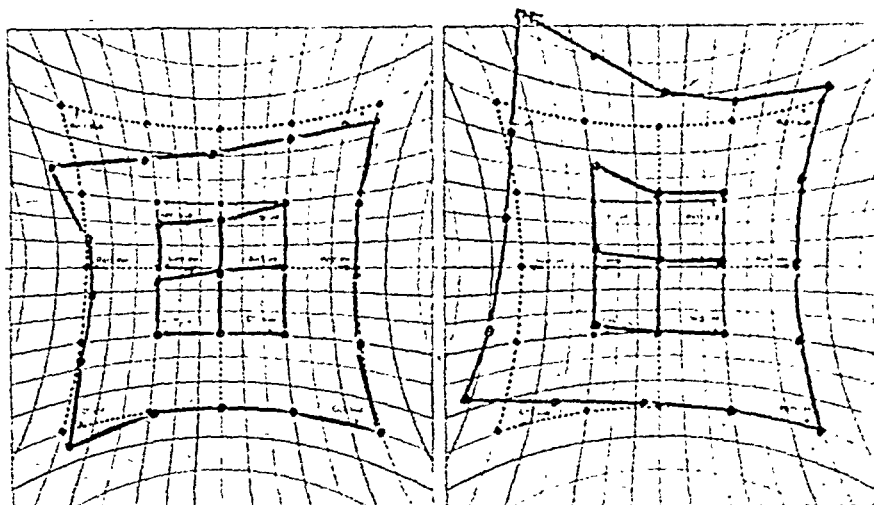
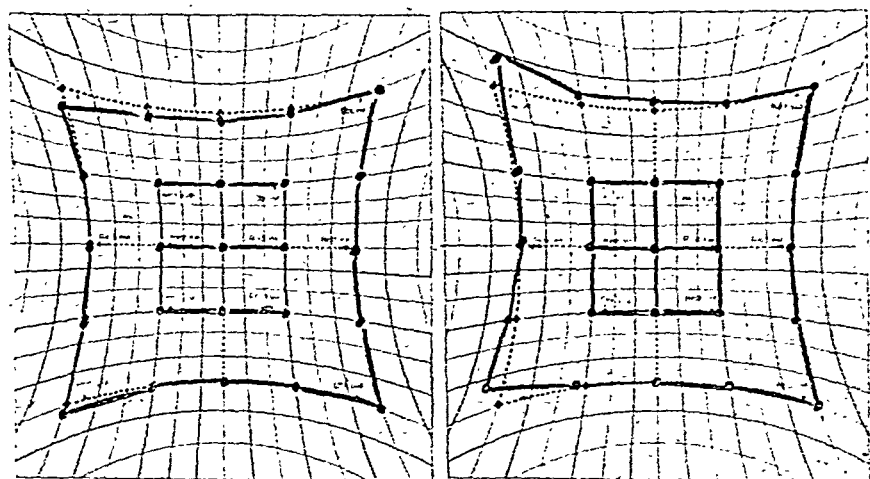


CASE 2.

PRE-OPERATIVE MUSC CHART.

Left Eye.
Paralysis of superior rectus

Right Eye.
Overaction of inferior oblique.

POST-OPERATIVE MUSC CHART.

Functionally Normal

CASE No. 2.

HISTORY.

J. C., aged 21 years. Pilot. On August 13, 1944, aircraft, of which he was pilot, crashed and burst into flames. Admitted to Burns Unit 1½ hrs. later with extensive burns and fractures. Injuries included depressed fracture of right malar, and a comminuted fracture of left external malleolus. Depression of left orbital floor shown later (v.i.). On September 28, 1944, burns well healed. Leg in plaster below the knee. Referred to ophthalmologist on account of a head tilt and diplopia.

PRE-OPERATIVE EXAMINATION. October 13, 1944.

Right malar fracture with obstruction of the right naso-lacrymal duct. Holds head over to the left.

Vision :—R 6/6. L. 6/6.

Movements :—Limited elevation of left eye, looking to left, with overshoot of right eye. Slight limitation of left external rectus.

Diplopia :—Vertical to left; lower image belonging to the right eye, increasing on elevation.

Maddox rod :—Esophoria 3–5 pd. R/L 10 pd.

Synoptophore :—S.P. angle—centre ... + 1° R/L 7–8 pd.

to right ... + 1° R/L 2–4 pd.

to left ... – 3° R/L 16–18 pd.

Fixing left eye slightly more than fixing right.

Fusion—0°. R/L 7° pd. Exyclophoria 4°.

Hess chart :—Right eye—overaction of right inferior oblique.

Left eye—paresis of left superior rectus.

Diagnosis :—Paresis of left superior rectus, left external rectus.

OPERATION. November 6, 1944.

Myectomy of the right inferior oblique, at the same time as a right dacryocystorhinostomy.

POST-OPERATIVE MEASUREMENTS.

Maddox rod :—R/L 2 pd.

Synoptophore :—S.P. angle—0° R/L 1 pd. 25° to left—R/L 4–5 pd.

Fusion—0°. No hyperphoria.

Adduction to + 30°.

Movements :—Much improved, see Hess chart No. 2.

DISPOSAL.

To leave. To be re-admitted to the Plastic Unit for further repair.

COMMENT. The clinical features of this case were at first masked by the extensive swelling caused by the burns. An X-ray photograph taken in the antero-posterior position revealed no fracture of the facial bones (picture 1). Stereoscopic pictures taken later in the vertico mental position, showed depression of the left orbital floor and separation of the fronto-malar suture (picture 2). It is of significance, therefore, that these fractures can be, and often are, missed unless stereoscopic X-ray photographs are taken in the vertico-mental position of the head.

CASE No. 11.

HISTORY.

C. C., aged 28 years. Instrument repairer II. On March 25, 1944, he stumbled over a tree in the blackout and a branch penetrated the lower lid of the right eye, passing backwards between the orbit and the globe. He was taken to a military hospital, where he was found to be suffering from ptosis of the right upper lid, proptosis due to severe retrobulbar haemorrhage, and absent eye movements. Some slow recovery took place and the vision improved to Right 6/9 with – 0.50 cyl 180° = 6/6. Left vision 6/6.

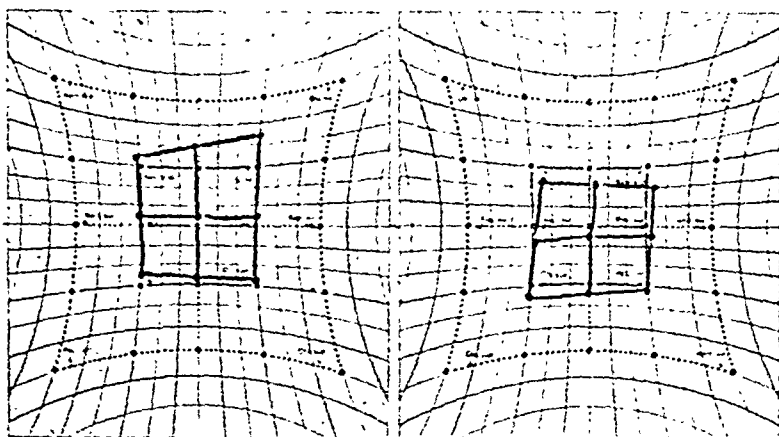
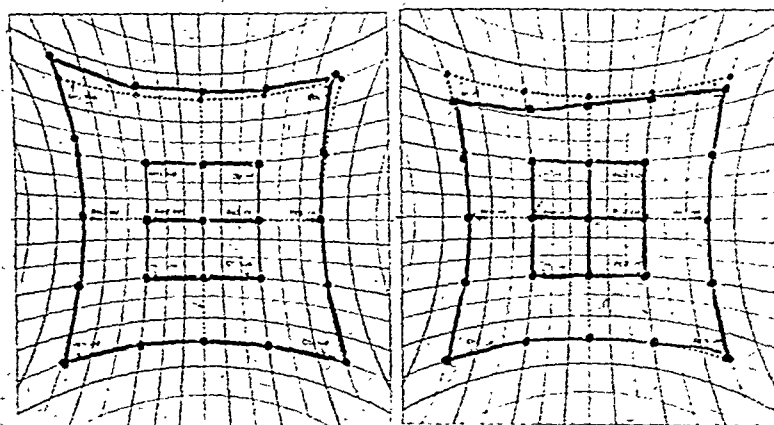
He was transferred to another military hospital where the following report was made :—

"As a result of a perforating wound of the orbit, this man appears to have damaged the right third and sixth nerves. I recommend a shade for the right eye and prolonged convalescence."

After a further month he was recommended for invaliding on account of intractable diplopia :—

"The lesion may well be permanent, and I think he should be discharged as unfit for further service."

CASE 11.

FIG. 1. PRE-TREATMENT.17th Nov 1944.Left Eye.Overaction of the elevator
muscles, particularly of the
inferior oblique.Right Eye.Excess of the elevator
muscles.FIG. 2. POST-TREATMENT.27th June 1944.Left Eye.Slight overaction
of the superior rectus.
(outer square only.)Right Eye.Very slight excess of
the inferior oblique
(outer square only.)

As a result of this pessimistic prognosis, this man was admitted to hospital on May 26, 1944, in a morbid and dejected condition to be, as he thought, invalided from the service. He was complaining of diplopia, mainly vertical in type, which had persisted unaltered since the time of his accident. He had suffered from no previous ocular abnormality and had never worn glasses.

EXAMINATION.

Vision :—R. 6/36—with no correction and much persuasion = 6/9.

L. 6/18—with + 0.50 sph.—1.0 cyl. at 180° = 6/9.

Movements :—Full.

Diplopia :—Vertical, maximum separation of images on laevo-elevation, the upper image belonging to the right eye.

Maddox rod :—Fixing right—L/R 3.5 pd. Exophoria 2 pd.

Fixing left—L/R 3.5 pd. Exophoria 1 pd.

Synoptophore :—S.P. angle—+1°. L/R 5 pd. 20° to right—L/R 7 pd.

Fusion—0° L/R 4 pd.

Infraduction 2 pd.

Adduction to 20° with effort.

Diagnosis :—Primary paresis of the right inferior oblique.

The notes of the case read, "this man is at present in a somewhat unco-operative state, he requires encouragement on the lines that he is going to get single vision, but must do his part to assist."

TREATMENT.

Orthoptic exercises.

At the end of the exercises he was sent out on a week's sick leave with the assurance that he would fully recover.

ON READMISSION. June 20, 1944.

Maddox rod :—L/R 1 pd. only.

Synoptophore :—S.P. angle—0°. L/R 2 pd.

Fusion—0°. No hyperphoria.

DISPOSAL.

Returned to duty.

Hess chart 1 :—Before treatment. R.E. Paresis of the elevator muscles.

L.E. Overaction of the elevators, particularly the inferior oblique.

Hess chart 2 :—After treatment. R.E. Very slight paresis of the inferior oblique.

L.E. Slight overaction of the superior rectus.

COMMENT. This record is given in full to illustrate the value of orthoptic exercises in the treatment of these cases.

Two months after an injury, this patient was complaining of a diplopia which had remained unaltered since the time of his accident. After a two weeks' course of orthoptic exercises his actual physical, as well as mental, outlook had completely altered, and the angle on the synoptophore had been reduced from L/R 8 to L/R 2.

It is not claimed that orthoptic exercises will cure a tropia; but they seem, as in this incidence, to light the path which leads to recovery, and supply the necessary stimulus to enable the patient to regasp and to hold with increased adduction strength, binocular single vision.

CASE No. 13.

HISTORY.

G. Y. S. Observer. On October 13, 1942, lacerated his left lower lid when he slipped and struck his head on a wire fence. The wound on his cheek turned septic and he says was followed by dizziness and fever. Now complaining of headaches and eye-strain after close work.

PRE-OPERATIVE EXAMINATION. February 18, 1944.

Vision :—R 6/6. L. 6/6.

Movements :—Limitation of right eye in dextro-elevation.

Diplopia :—Vertical, with maximum separation of images in dextro-elevation, the upper image belonging to the right eye.

Maddox rod :—Fixing right and left ... 2 pd. L/R.

Fixing right ... 2 pd.

Fixing left ... 1 pd.

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Synoptophore :—S.P. angle— -1° . L/R 1 pd., increasing to L/R 2 pd. 25° to right, with exocyclophoria.

Fusion -0° .

Adduction -10° .

Abduction -5° .

Hess chart :—Right eye—paresis of right superior rectus.

Left eye—overaction of left inferior oblique.

Diagnosis :—Paresis of right superior rectus.

OPERATION. March 6, 1944.

Left inferior oblique tenotomy.

POST-OPERATIVE MEASUREMENTS.

Maddox rod :—L/R 2 pd.

Synoptophore :—S.P. angle— 0° . L/R 2 pd. 20° to right—L/R $2\frac{1}{2}$ pd.

Fusion -0° .

Adduction $+20^{\circ}$, the L/R which increased with further adduction to L/R 8-9 pd.

OPERATION. April 4, 1944.

Division of adhesions round inferior oblique of left eye.

POST-OPERATIVE MEASUREMENTS. April 10, 1944.

Movements :—Full. No diplopia.

Maddox rod :—Esophoria 1 pd. L/R 1 pd.

Synoptophore :—S.P. angle— 0° . L/R $1\triangle$ in all positions.

Fusion— 0° . Supraduction to R/L 7 pd.

Adduction $+35^{\circ}$.

Abduction -5° .

Hess chart :—No muscle imbalance on inner or outer chart.

DISPOSAL.

To Medical Board. Returned to flying duties.

CASE No. 15.

HISTORY.

S. W. B., aged 25 years. Pilot. Returning from operational trip in a Typhoon (August 25, 1943) he was caught in the slip stream of the preceding machine, which, coming in a little high, elected to go round the airfield again, the port wing stalled and the Typhoon cartwheeled. Concussed for 16 hours with islets. Discharged from Army hospitals September 15, 1943, and admitted to an R.A.F. hospital October 7, 1943, suffering from (1) deep scarring of right upper lid at site of laceration, and (2) diplopia; this had been noticed since the time of the accident.

Now finds diplopia very troublesome when reading or walking downstairs.

PRE-OPERATIVE EXAMINATION.

Vision :—Right—6/9 with -0.75 cyl. at 180° —6/6.

Left—6/5.

Movements :—Limitation of depression of right eye, especially on laevo-depression.

Upper lid remains retracted at the inner angle on looking down.

Diplopia :—On laevo-version increasing on depression, the lower image belonging to right eye.

Maddox rod :—Exophoria 1 pd. R/L 2 pd.

Synoptophore :—S.P. angle -3° . R/L 1 pd., increasing to $2\frac{1}{2}$ pd. to left.

Adduction $+8^{\circ}$.

Abduction -5° .

Hess chart 1 (October 13, 1943) :—Right eye—shows paresis of right superior oblique.

Left eye—shows overaction of left inferior rectus.

Diagnosis :—Paresis of right superior oblique, due to injury and scarring round the trochlea.

OPERATION. November 18, 1943.

Scar on upper lid excised and an attempt made to free the trochlea. One week's orthoptic treatment followed.

POST-OPERATIVE EXAMINATION.

Diplopia :—Not improved.

Maddox rod :—Exophoria 2 pd. R/L 4 to 6 pd. (worse than before). Re-admitted December 31, 1943, for a skin graft to right upper lid.

EXAMINATION ON April 2, 1944.

Maddox rod :—Fixing right—R/L 6 pd. Esophoria 1 pd.

Fixing left—R/L 10 pd. Esophoria 2 pd.

Synoptophore :—Fixing right—R/L 8 pd., increasing to left.

Fixing left—R/L 5 pd., increasing to left.

June 30, 1944. Injured by a flying bomb in London. He sustained (1) rupture of tympanic membranes, (2) concussion, and (3) laceration of scalp. After recovering from these injuries he was re-admitted to hospital October 3, 1944.

PRE-OPERATIVE EXAMINATION. October 5, 1944.

Double vision has remained constant during the past five months, now fourteen months since his crash. Slight right enophthalmos.

Vision :—Right—6/5 with +0.25 sphere—6/5.

Left—6/5 with +0.25 sphere—6/5.

Movements :—Limitation of depression of right eye, especially to the left.

Diplopia :—Corresponds to paresis of right superior oblique muscle—as before.

Maddox rod :—Fixing right—R/L 4 pd., no lateral deviation.

Fixing left —R/L 6 pd., no lateral deviation.

Slight downward movement of head makes deviation R/L 12 pd.

Synoptophore :—S.P. angle—fixing right—centre ... 0° R/L 4 pd.

25° to right ... 0° R/L 2.5 pd.

25° to left ... 3° R/L 26 pd.

fixing left —centre ... 0° R/L 3½ pd.

25° to right ... R/L 2 pd.

25° to left ... 3° R/L 23 pd.

right exocyclophoria 2°.

Fusion angle—0° R/L 4 pd., no infraduction.

Adduction to 25°.

Abduction to -4°.

Hess chart 2 :—Worse than before, with Inhibitional palsy L. S. Rect.

B.H.T.D. :—(P.D. 60) R/L 20 = (Bishop Harman Diaphragm Test).

OPERATION. October 23, 1944.

Recession of left inferior rectus 3 mm.

POST-OPERATIVE MEASUREMENTS. October 31, 1944.

Maddox rod :—Fixing right—R/L 1 pd.

Fixing left —R/L 1 pd.

Maddox wing :—Orthophoric.

Synoptophore :—S.P. angle—centre ... -2° L/R 1 pd.

25° to right ... L/R 1.5 pd.

25° to left ... R/L 1.5 pd.

Fusion—0. No hyperphoria.

Adduction to +35°.

Abduction to -5°.

B.H.T.D. :—(P.D. 60) R/L at 4.

Maddox rod :—Increased to R/L 2-3 pd., after 14 days, but eyes remained comfortable and patient could read without difficulty.

Diplopia :—Experienced only at extreme of vision down to left and up to right.

MEASUREMENTS. November 16, 1944.

Maddox rod :—Exophoria 1 pd.—fixing right—R/L 3 pd.

fixing left —R/L 2 pd.

Diplopia :—R/L on laevo-depression.

Synoptophore :—S.P. angle—fixing right—centre ... 0° R/L 3-4 pd.

to left ... R/L 7 pd.

fixing left —centre ... 0° R/L 1-2 pd.

to left ... R/L 5 pd.

Fusion—0°, no hyperphoria to sides.

Adduction to +45°.

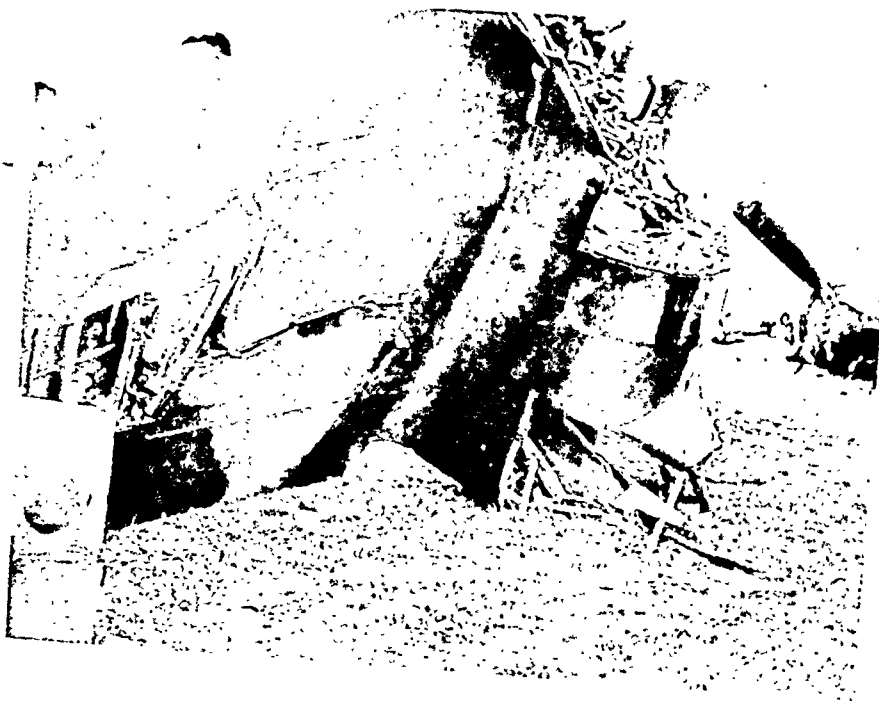
Abduction to -5°.

Hess chart 3 :—Very good.

Eyes very comfortable and can read without difficulty.

DISPOSAL.

To Medical Board. Repatriated to Australia.



CASE 15.

Extensive wreckage of Typhoon from which the pilot emerged with nothing worse than concussion and diplopia, from which he made a complete recovery.

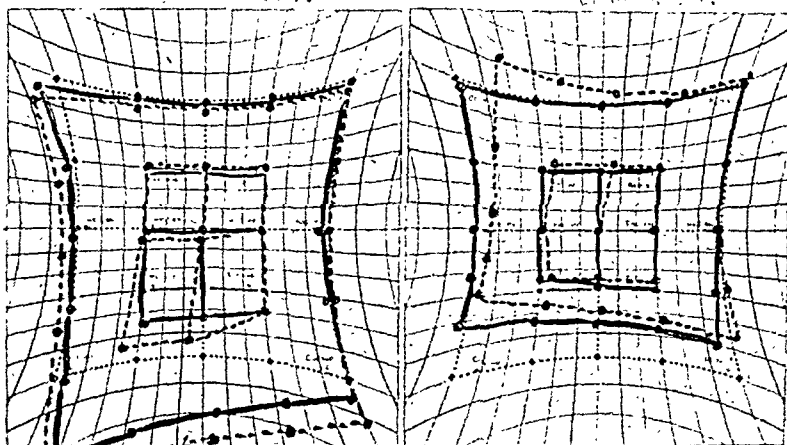
Left Eye
overaction of inferior
rectus

1935 OCT. 20, 1940

Wess Guit No. 2
(dotted lines on right eye)

Overaction of inferior
rectus, the initial and first
of the inferior rectus

Wess Guit No. 2
initial



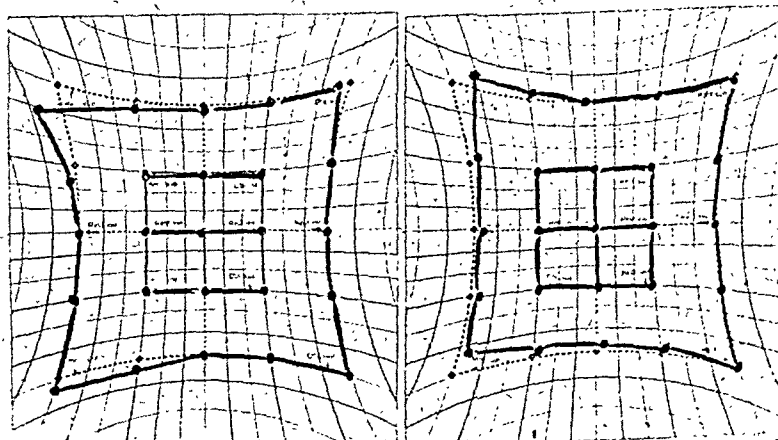
Wess Guit No. 2

Left Eye

Wess Guit No. 2

Inner overaction of
Outer overaction
initially inferior
superior rectus
extreme range of movement

Wess Guit No. 2
initially inferior
superior rectus
extreme range of movement



CASE 18—BEFORE OPERATION



AFTER OPERATION



CASE No. 18.

HISTORY.

A. C. H., aged 22 years. Pilot—under training. Crashed in Canada in August 1942, when the navigator fainted and fell across the controls. He sustained a compound fracture of the frontal bone; this was followed by a cerebral abscess which was drained in Canada. He noticed diplopia after the operation. On returning to England, two operations were performed, for repair of his frontal bone.

PRE-OPERATIVE EXAMINATION. October 27, 1943.

Marked head tilt down and to the left.

Vision :—R 6/5. L. 6/5.

Movements :—Limitation of right eye on laevo-depression and laevo-version.

Diplopia :—Vertical. Maximum separation of images on laevo-depression, the lower image belonging to the right eye.

Maddox rod :—Fixing right—Exophoria 1 Δ . R/L 15 pd.

Fixing left —Exophoria 1 Δ . R/L 12 pd.

Synoptophore :—S.P. angle—fixing right—centre ... 0° R/L 8 pd.

20° to right ... R/L 4 pd.

20° to left ... R/L 15 pd.

fixing left —centre ... R/L 6 pd.

20° to right ... R/L 3 pd.

20° to left ... R/L 14 pd.

Fusion—0°. No hyperphoria straight ahead.

Adduction +25°.

Abduction —4°.

Hess chart :—Right eye—paresis of right superior oblique, secondary overaction of right inferior oblique.

Left eye—overaction of left inferior rectus.

Diagnosis :—Paresis of right superior oblique.

OPERATION. January 12, 1944.

Right inferior oblique myectomy.

POST-OPERATIVE MEASUREMENTS.

Maddox rod :—No horizontal deviation. R/L 4 pd.

Synoptophore :—S.P. angle—0° R/L 4 pd. increasing to R/L 5 pd. to left.
decreasing to R/L 3 pd. to right.

Fusion—0°.

Adduction +30°.

Abduction —5°.

Supra-duction L/R 4 pd.

DISPOSAL.

To ground duty.

CASE No. 30.

HISTORY.

J. R. D., aged 23 years. Flight engineer. Aircraft crashed and caught fire on June 7, 1944. He sustained burns to right arm, wrist, face and neck. Had a period of amnesia of $\frac{1}{2}$ hour before, and 3 hours after the accident. Pilot was severely wounded, air gunner died. He complained of diplopia immediately he could concentrate after the accident. X-ray of skull showed no fracture. Thought to be hysterical.

PRE-OPERATIVE EXAMINATION. September 2, 1944.

Vision :—R. 6/6 with +0.75 sphere = 6/5.

L. 6/6 with —0.25/+0.50 cyl. at 85° = 6/5.

Movements :—Weakness of external recti and over elevation of left eye on dextro-version.

Diplopia :—Homonymous at all distances over 1 foot, with left hyperphoria increasing to right.

Maddox rod :—Esophoria greater than 30 pd. Left hyperphoria 5 pd.

Synoptophore :—S.P. angle—fixing right—centre ... +18° L/R 6 pd.

to right ... L/R 10 pd.

to left ... L/R 1 pd.

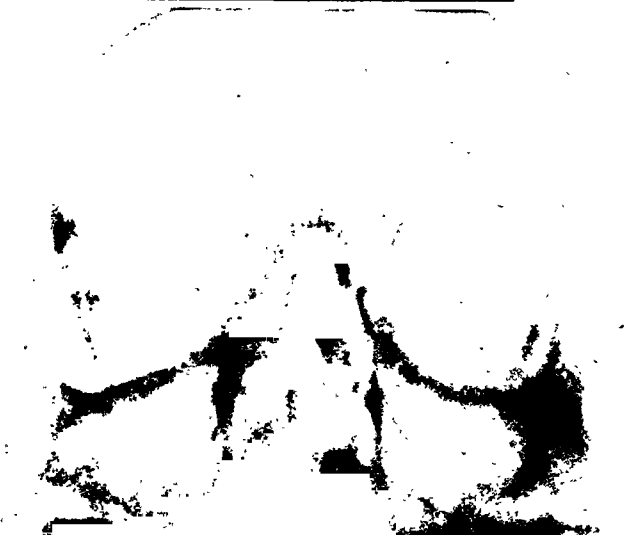
fixing left —centre ... +15° L/R 2 pd.

to right ... L/R 5 pd.

to left ... L/R 4 pd.

CASE 18

Compound fracture of right frontal bone



1933-1937

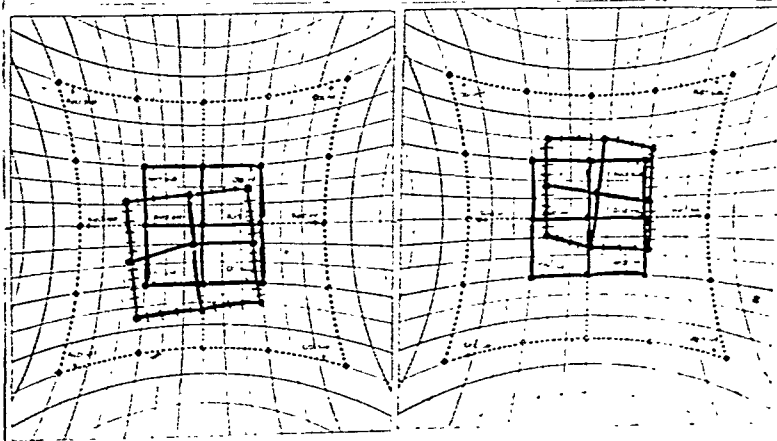
Before operation (++++)
After operation (—)

Left eye

Distraction of the inferior
rectus (++++)
Orthophoric (—)

Right eye

Paralysis of the superior
oblique (++++)
Orthophoric (—)



CASE 52

BEFORE OPERATION.

AFTER OPERATION.

Over elevation of right eye
with diplopia .

No over elevation of right eye
no diplopia .



FRACTURE OF LEFT MAXILLA: OPAQUE ANTRUM .
Wiring of frontomalar suture .

Fusion $\div 15^\circ$. L.R 3 pd.

Adduction to 28° with increase of left hyperphoria.

Abduction to -10° .

Hess chart :—Bilateral paresis of external recti—right more than left.

Diagnosis :—As Hess chart above.

September 26, 1944. Improvement with exercises. Now no diplopia for close objects at about 3 feet. Had 14 days leave.

Synoptophora (October 11, 1944):—S.P. angle \div S. L/R 4 \triangle —5 \triangle straightening
to \div 2 pd.

Maddox rod (November 6, 1944):—Still shows esophoria 10-25 pd.

OPERATION. November 8, 1944.

Recession of right internal rectus.

POST-OPERATIVE MEASUREMENTS. November 12, 1944.

Synoptophore :—Angle much the same as before.

OPERATION. November 15, 1944.

Tenotomy of left inferior oblique.

POST-OPERATIVE MEASUREMENTS. November 21, 1944.

Maddox rod :—Esophoria S Δ I. R 2 pd.

CASE 30

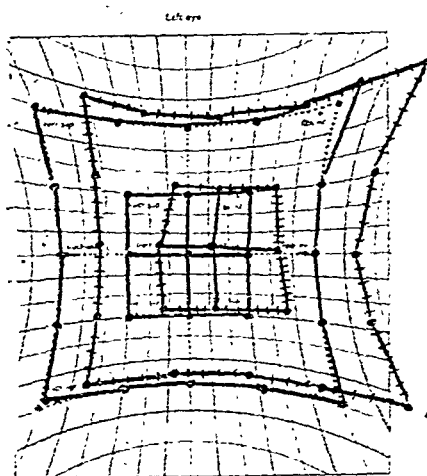
ALL INFORMATION CONTAINED HEREIN IS UNCLASSIFIED

(the right more than the left).

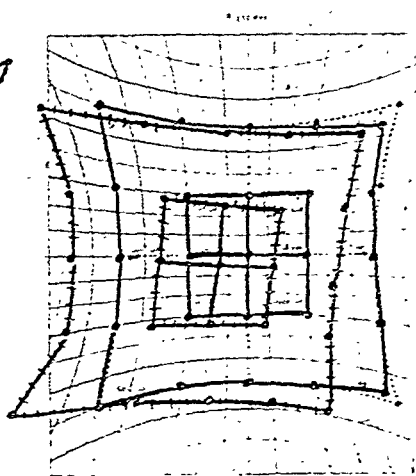
RES. 247.

REPOF operation, (= ~~+++~~)

After operation, 12-1-1



Green glass in front of left eye



Green & red in front of right eye

J. C. NEELY

Synoptophore :—S.P. angle $+3^{\circ}$ - 4° L/R 1 pd. 25° to right—L/R 3 pd.
 Fusion— 0° . No hyperphoria. Holds to sides.
 Adduction to $+25^{\circ}$.
 Abduction to -6° .

Hess chart :—Very good indeed.

DISPOSAL.
 To Central Medical Board. Passed fit for flying duties.

CASE No. 34.

HISTORY.

R. W., aged 29 years. Instrument repairer. Accident on July 25, 1942. Thrown over handlebars of bicycle. Noticed double vision on recovering consciousness. It is reported that there was definite evidence of severe damage to the central nervous system—weakness of limbs in all movements to the right. Diplopia was at first present in all directions, now has single binocular vision on looking about 20 to the left, otherwise the vertical diplopia is still present, and increases on looking to the right. Says he has learnt to put up with it fairly well; as he concentrates on the higher image. Has very marked head position; head down and chin towards the right shoulder.

PRE-OPERATIVE EXAMINATION. November 22, 1942.

Vision :—R. 6/6. L. 6/6.
Movements :—On dextro-version the left eye deviates upwards.
 On dextro-depression—defective depression of left eye.
 On dextro-elevation—defective elevation of right eye with over-elevation of left eye.

Diplopia :—Vertical. Maximum separation down and to the right, the lower image belongs to the left eye. There is also tilting of the image seen by the left eye inwards (excyclo).
Maddox rod :—Fixing right—Exophoria 3 pd. L/R 12 pd.
 Fixing left—No lateral deviation. L/R 14 pd. very variable and some excyclophoria.

Synoptophore :—S.P. angle—centre ... 1° L/R 15 pd., excyclophoria 10° - 12° .
 20 to right ... 1° L/R 10 pd. excyclophoria 10° .
 20 to left ... 1° L/R 2 pd. excyclophoria 2° .
 —centre ... 0° L/R 10 pd. excyclophoria 10° , very

Fusion
 little infraduction.

Side movements impossible on account of incomitance.
 Adduction to $+5^{\circ}$.
 Abduction to -2° .

Hess chart :—Shows paresis of the left superior oblique.
 Shows paresis of the right superior rectus.
 Shows overaction of right inferior oblique.
 Shows overaction of the left superior oblique. Secondary paresis of the right superior rectus.

Diagnosis :—Paresis of the left superior oblique.
 superior rectus.

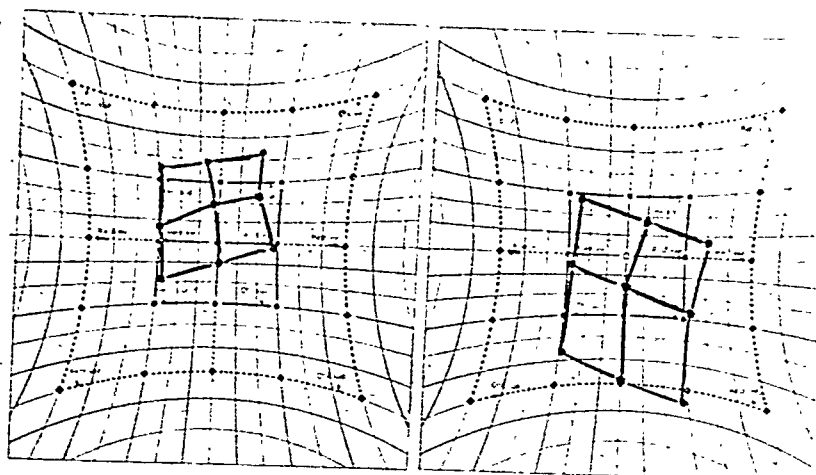
OPERATION. November 25, 1942.
 Retroplacement of the right inferior rectus 2-3 mm.

POST-OPERATIVE MEASUREMENTS.
Maddox rod :—Fixing right—Esophoria 1Δ L/R. 6 pd.
 Fixing left—No lateral deviation. L/R 9 pd.

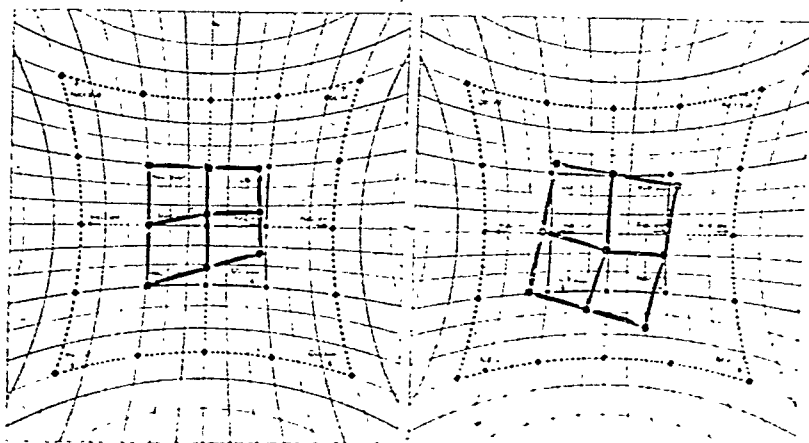
Synoptophore :—S.P. angle—centre ... L/R 3 to 6 pd.
 20 to right ... L/R 12 pd.
 20 to left ... L/R 2 pd.

Fusion— 0° L/R variable 0 to 6 pd.
 Adduction to $+15^{\circ}$.
 Abduction to -4° .

Some improvement after operation, but still considerable diplopia to the right.
 OPERATION. December 10, 1942.
 Myectomy of the left inferior oblique.



Hirsch Chart.
After First Operation.



J. C. NEELY

CASE 34.

FIGURE 1.
STEP SECOND OPERATION.

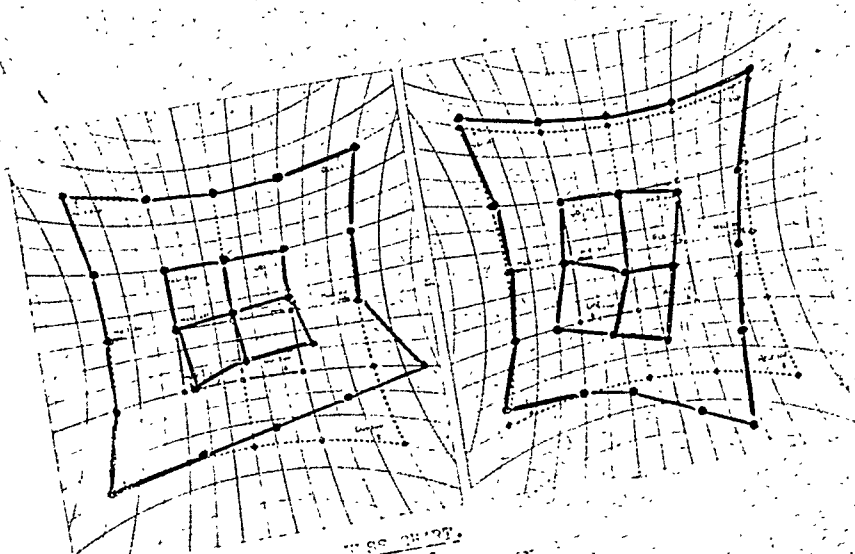
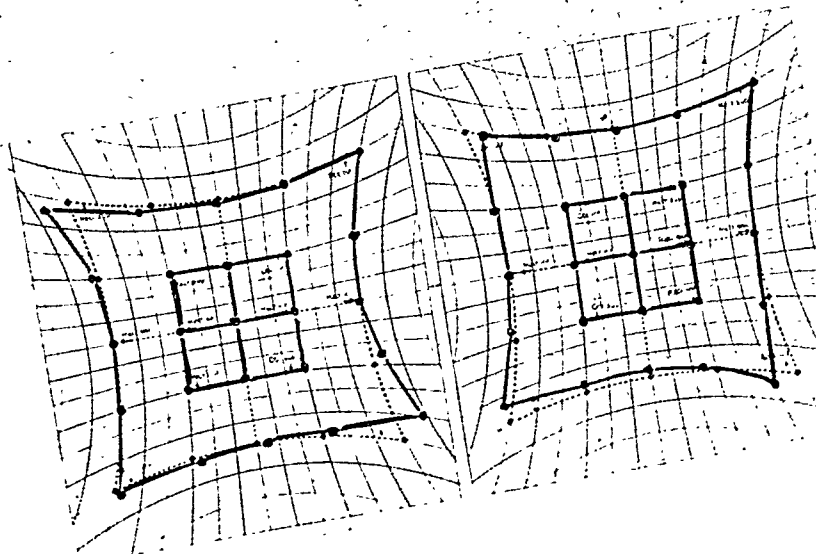


FIGURE 2.
STEP THIRD OPERATION.



Diplopia :—Present on laevo-version and depression; maximum displacement at the latter position, the more displaced image belonging to the right eye.

Maddox rod :—Exophoria 4 pd. R/L 6 pd.

Synoptophore :—S.P. angle—centre ... -1° R/L 1.5 pd.

20° to left ... R/L 3.5 pd.

Fusion -1° R/L 1.5 pd.

Adduction to $+35^{\circ}$.

Abduction to -4° .

No infraduction.

Hess chart :—Right eye—paresis of right superior oblique.

Left eye—overaction of left inferior rectus.

Diagnosis :—Paresis of right superior oblique.

On August 28, 1944, on return from 14 days sick leave, the right hyperphoria had decreased to 1 pd. Fusion was maintained to the left, and he felt that his eyes were still improving.

DISPOSAL.

No operation advised. Return to unit for non-flying duties for two months.

COMMENT. It was considered by the neurological specialists that the intramedullary haemorrhage could be properly attributed to the head injury which this navigator sustained on leaving the aircraft, although it was unusual for symptoms such as these to be delayed for so long.

Orthoptic treatment was given both at the military hospital and again at the R.A.F. hospital when the patient was transferred. From a right hypertropia of 12–16 pd., with constant diplopia the muscle imbalance had decreased to R/L 1 pd. in three months.

CASE No. 49.

HISTORY.

G. W. C., aged 24 years. Fitter, under training as Flight Engineer. Head injuries (i) in 1938 playing football, unconscious 3 to 4 days, (ii) in 1942 at football, unconscious 1 hour, (iii) in 1943 playing ice hockey, unconscious 12 hours. After last accident he was transferred to hospital for eye treatment.

PRE-OPERATIVE EXAMINATION. August 21, 1943.

Vision :—R. 6/5. L. 6/5.

Movements :—On looking to the left, the right eye turns up and the left eye turns down.

Diplopia :—Vertical straight ahead, increasing to the left; maximum separation of images on laevo elevation, upper image belonging to the left eye.

Maddox rod :—Fixing right and left—Esophoria $\frac{1}{2}$, R/L 8 pd.

Synoptophore :—S.P. angle—fixing right—centre ... 0° R/L 13 pd.

to right ... 0° R/L 4–6 pd.

to left ... 0° R/L 22 pd.

fixing left —centre ... 0° R/L 7–12 pd.

to right ... 0° R/L 5 pd.

to left ... 0° R/L 24 pd.

Measurements were variable and unsteady. Marked suppression left eye. Suppressed left eye on fusion.

Hess chart :—Suppressed too much, initially, for charting, but after 14 days orthoptic treatment shows :—

Right eye—paresis of right superior oblique; overaction of inferior oblique.

Left eye—paresis of superior rectus; overaction of inferior rectus.

Diagnosis :—Paresis of left superior rectus with overaction of the right inferior oblique and secondary palsy of the right superior oblique.

FIRST OPERATION. September 13, 1943.

Myectomy of right inferior oblique.

POST-OPERATIVE MEASUREMENTS.

Synoptophore :—S.P. angle—fixing right—centre ... -2° R/L 4 pd.

to right ... -2° R/L 2 pd.

to left ... -2° R/L 16 pd.

fixing left —centre ... -2° R/L 4 pd.

to right ... -2° R/L 2 pd.

to left ... -2° R/L 17 pd.

Fusion momentarily with no hyperphoria; no ductions.

CASE 49.

HESS CHART No. 1.POST-OP. FIRST OPERATION
SEPTEMBER 20, 1943. (-----)Left Eye

Intropia of the
superior rectus.
Overaction of the
inferior rectus.

Right Eye

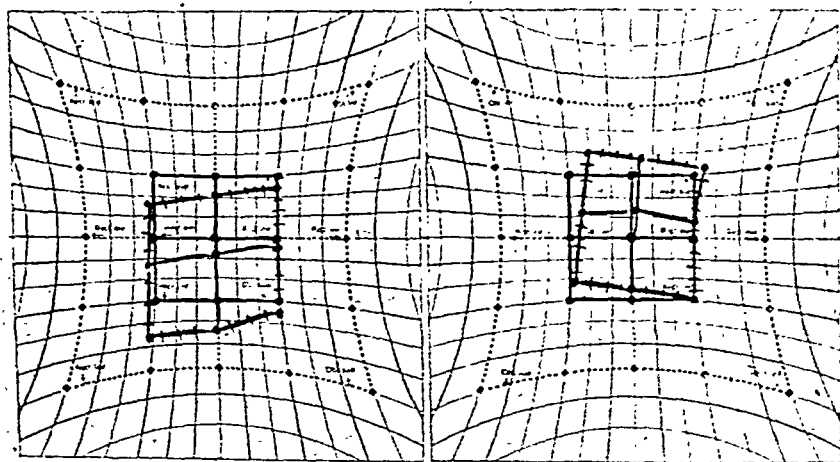
Exotropia of the
superior rectus.
Overaction of the
inferior rectus.

HESS CHART No. 2.POST-OP. SECOND OPERATIONSEPTEMBER 21, 1943. (-----)Left Eye

No muscle imbalance
inner chart.

Right Eye

No muscle imbalance
inner chart.



Diplopia :—On looking upwards and to the left with exocyclophoria
Hess chart 2 (September 20, 1943).

SECOND OPERATION. September 21, 1943.

Recession of left inferior rectus—3 mm.

POST-OPERATIVE MEASUREMENTS.

Maddox rod :—L/R 9 pd (over-correction).

Synoptophore :—S.P. angle—centre ... 0° L/R 6-7 pd.

to right ... 0° L/R 9 pd.

to left ... 0° L/R 1 pd.

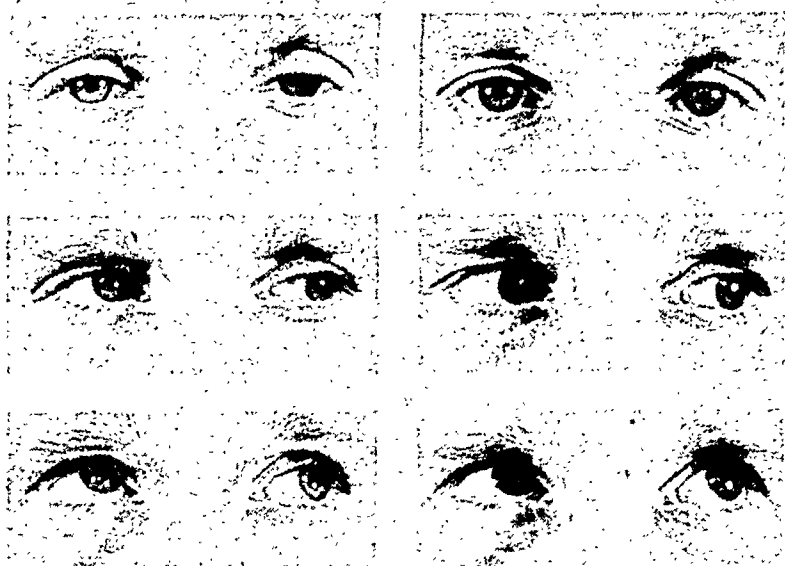
Fusion—0° L/R 7 pd.

Supraduction to L/R 4 pd.

CASE 49

BEFORE OPERATION.
Over elevation of right eye

AFTER OPERATION.
Eyes level, no over elevation



MEASUREMENTS AFTER THREE WEEKS SICK LEAVE.

Maddox rod :—Exophoria 1 pd. L/R 1-2 pd.
Synoptophore :—S.P. angle—centre ... L/R 1 pd.
 to right ... L/R 1-2 pd.
 to left ... L/R 2-3 pd.
 Fusion—0°. No hyperphoria.
 Adduction +15°.
 Abduction -5°.

Hess chart 3 :—No muscle imbalance; inner chart (October 15, 1943).

On October 23, 1943, discharged to duty.

Re-admitted to hospital on January 13, 1944. Diplopia has recently recurred. After 2 hours close work the image seems to tilt and separate. Diplopia most marked to the right, showing slight overaction of the left inferior oblique.

EXAMINATION.

Maddox rod :—Fixing right—Exophoria $\frac{1}{2}$ Δ , L/R 1 pd.
 Fixing left—Exophoria $\frac{1}{2}$ Δ , L/R 1-2 pd.
Synoptophore :—S.P. angle—L/R 1 pd.—decreasing to 0 to the left,
 increasing to 2 Δ -3 Δ to the right.
 Fusion—0°. L/R 2 pd. Excyclophoria 2°.

THIRD OPERATION. January 21, 1944.

Partial tenotomy of left inferior oblique.

POST-OPERATIVE MEASUREMENTS. January 25, 1944.

Maddox rod :—Exophoria 1-2 pd. L/R 1 pd.
Synoptophore :—S.P. angle—1°-2°. L/R 1-2 pd.

RE-EXAMINED—April 29, 1944.

Maddox rod :—Orthophoric, laterally—L/R $\frac{1}{2}$ -1 pd.

Synoptophore :—S.P. angle ... 0° L/R $1\frac{1}{2}$ pd.

Fusion ... 0° L/R $\frac{1}{2}$ pd.

Ocular movements full, no diplopia.

DISPOSAL. To Medical Board. Taken off flying on account of his head injuries, and not for ocular reasons.

CASE No. 52.

HISTORY.

A. P. C., aged 22 years. Pilot. Crashed on October 21, 1942, in a glider. His face was lacerated and left maxilla fractured. The fronto-malar suture was wired at hospital. He noticed double vision immediately after the accident. The diplopia has improved and now has single vision below eye level. First seen three months after the accident, when there was some ptosis of the left upper lid, enophthalmos and displacement of the left eye downwards. Admitted to hospital again five months after the accident for treatment of his diplopia.

PRE-OPERATIVE EXAMINATION.

Stereoscopic films showed that the position of the floor of the left orbit was satisfactory.

Vision :—R. 6/6. L. 6/6.

Movements :—Defective elevation of the left eye. Over elevation of the right eye.

Limitation of internal rotation of left eye.

Diplopia :—Vertical above eye level; the higher image belonging to the left eye.

Little difference to right and left. Horizontal. Crossed diplopia to the right.

Maddox rod :—Fixing right—Exophoria 2°. R/L 10-16 pd.

Fixing left —Exophoria 3°. R/L 5 pd.

Synoptophore :—S.P. angle—fixing right and left—centre ... R/L 9-11 pd.
to right ... -5, R/L 15 pd.
to left ... R/L 7 pd.

to right—excyclophoria 10°.

to left excyclophoria 3°.

Fusion angle—0°, R/L 7 pd., excyclophoria 3 pd.

Adduction—poor, $\div 10^\circ$ (approximately).

Abduction to -2° .

Hess chart 1 :—Left eye—paresis of elevators.

Right eye—overaction of elevators.

Diagnosis :—Left third nerve paralysis with partial recovery and residual paresis of left superior rectus, left inferior oblique, and left internal rectus.

OPERATION.

Recession of right superior rectus. Tenotomy of right inferior oblique.

POST-OPERATIVE MEASUREMENTS.

Maddox rod :—Orthophoric.

Diplopia :—No diplopia at 30° above eye level. Some R/L on further elevation.

For this he compensates by slightly raising his head.

Synoptophore :—S.P. angle—centre ... 0° R/L 1.5 pd.

to right ... -5° , R/L 5 pd.

to left ... $+4^\circ$, R/L $\frac{1}{2}$ pd.

Fusion—0°. No hyperphoria holds to 25 to right or left.

Adduction to $+25^\circ$.

Adduction to -3° .

Stereoscopic vision full.

Hess chart 2 :—No muscle imbalance on inner chart.

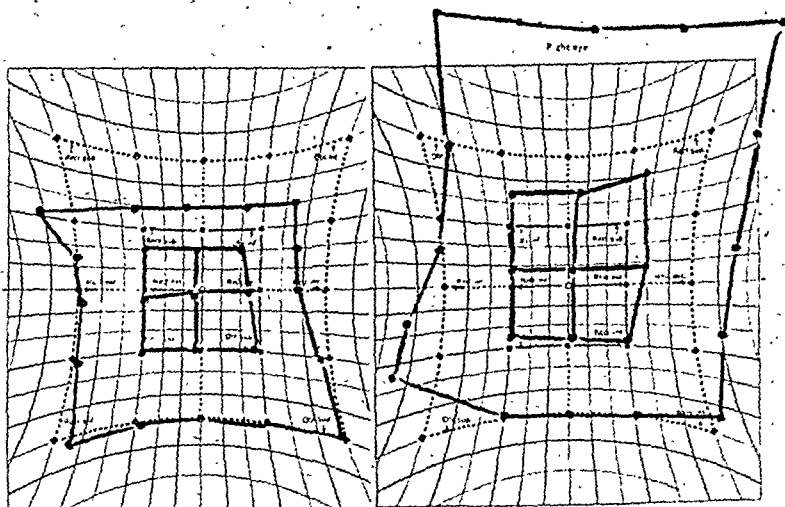
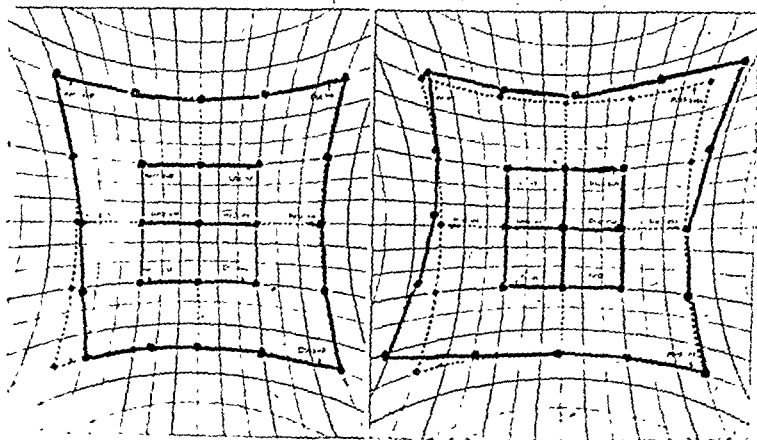
DISPOSAL. To Central Medical Board, where he was passed fit for limited (i.e. non-operational) flying.

CASE No. 53.

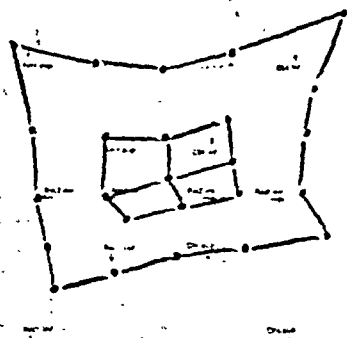
HISTORY.

F. M., aged 23 years. Pilot. Aircraft crashed over Germany in November, 1941. P.O.W. in Stalag Luft III for three years. He was badly concussed and saw double from the time of regaining consciousness. There has been no improvement in the diplopia; he has adopted a marked head tilt downwards and to the right. Now repatriated; admitted to hospital on account of the constant diplopia.

CASE 52

PRE OPERATIVE HESS CHART.Paralysis of left elevator muscles.Overaction of right elevator muscles.POST OPERATIVE HESS CHART.

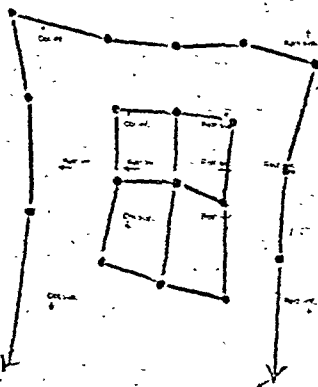
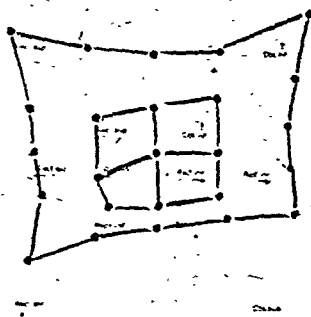
CASE 53

Case 53.Before 1st. operation. 10.4.46.Left eye.
Laxity of superior oblique.Right eye.
Overaction of inferior rectus.
Secondary laxity of primary rectus.

Green glass in front of right eye

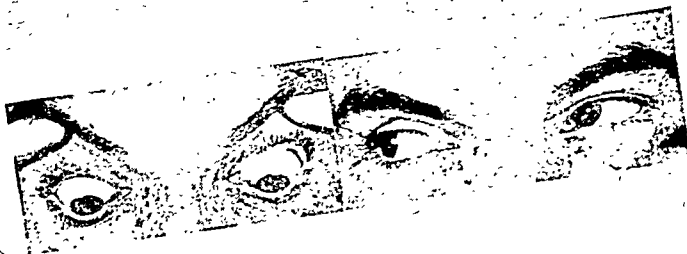
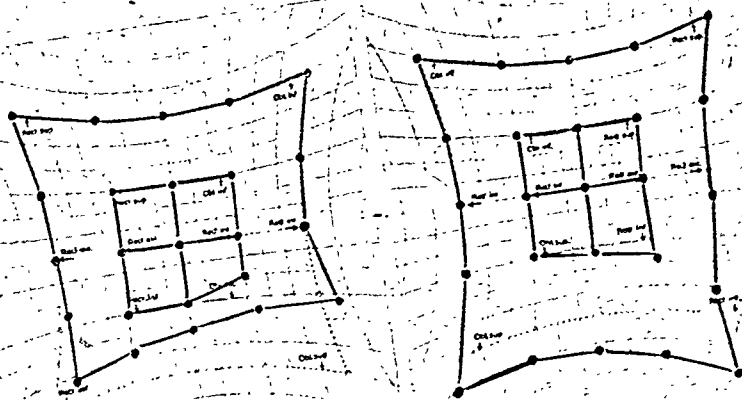
Second. operation. 1.11.46.

Myotomy of left inferior oblique. Recession of right inferior rectus.



after 2nd. and 3rd. operation.

2) Complete tenotomy of right inferior rectus. (1) tenotomy of left inferior oblique.



PRE-OPERATIVE EXAMINATION. April 17, 1945.

Vision :—R.E. 6/6 with + 1.0 sph = 6/4.
L.E. 6/12 with + 1.0 sph/+ 1.25 c 145° = 6/6.

Movements :—Defective depression of L.E. and overaction of R.E. to right, marked overaction of L.E. on elevation.

Diplopia :—Vertical. Maximum displacement of images on dextro-depression and laevo-elevation.

Maddox rod :—Controlled, Esophoria 7Δ L/R 7Δ-8Δ. Relaxed, L/R 32Δ-35Δ.
Synoptophore :—S.P. centre ... +4° L/R 8Δ. +4° L/R 26Δ Exyclo 6
To right ... L/R 26Δ. L/R ++
To left ... L/R 4Δ Exyclo. 4°. L/R 14Δ.

Fusion +3°, L/R 7Δ, Exyclo 5°
Adduction -10°. Abduction -1°.

Hess chart :—L.E.—Paralysis of left superior oblique; overaction of left inferior oblique. R.E.—Paresis of right superior rectus; overaction of right inferior rectus.

Diagnosis :—1. Concussion.
2. Paralysis of left superior oblique.

OPERATION. May 2, 1945.

Myectomy of left inferior oblique. Recession of right inferior rectus.

POST-OPERATIVE MEASUREMENTS. May 14, 1945.

Maddox rod :—F.R. Esophoria 5Δ L/R 3Δ .

F.L. Esophoria 7Δ L/R 4Δ — 5Δ .

Ocular Movement :—Good, up and to right, but marked overaction of R.E. on depression.

Synoptophore :—S.P.—centre ... $\pm 4^\circ$, L/R 2Δ Encyclo 4° .

to right ... $\pm 4^\circ$, L/R varying to 14Δ .

to left ... $\pm 4^\circ$, L/R 1Δ to 1Δ .

Fusion $\pm 4^\circ$, L/R 2Δ breaking to R.

Adduction $\pm 8^\circ$. Abduction to -1° .

RE-ADMITTED—May 30, 1945.

Measurements substantially the same.

OPERATION—June 6, 1945.

Complete tenotomy and freeing of right inferior rectus.

POST-OPERATIVE MEASUREMENTS. June 6, 1945.

Unimproved.

CASE 54.

RIGHT 6th. NERVE PALSY.



Subject looking to the right, shewing:—

- 1) Complete paralysis of right external rectus, with overaction of left internal rectus, prior to operation.
- 2) 15° binocular movement to right, following "O'Connor's" operation.

OPERATION. July 13, 1945.

Tenotomy of left inferior oblique at the globe.

POST-OPERATIVE MEASUREMENTS. July 17, 1945.

Synoptophore :—Angle—centre ... +2°
 20° to R. ... L/R 4 Δ.
 20° to L. ... L/R 3 Δ.

Fusion 0. Adduction to +20°. Abduction to 2°.

ON RELEASE FROM THE SERVICE.

There was no diplopia ahead or to the left, some diplopia on looking down and to the right, for which he compensated by bending his head. Movements were good and head held much straighter.

Hess chart :—No. 3 shows the comparatively small asymmetry confined to the lower right quadrant. It is perhaps important to note that (1) this officer had been a P.O.W. for three years, during which time he had received no treatment for his diplopia.

(2) That the recession and subsequent tenotomy of the right inferior rectus, reduced but did not completely eliminate the overaction of this muscle.

(3) That a generous tenectomy of the left inferior oblique, at its origin, similarly only reduced its overaction.

(4) That a tenotomy of the inferior oblique near its insertion to the globe, reduced the vertical height by another 10 P.D. in the line of action of the muscle, to render the subject orthophoric ahead and to the left.

CASE No. 55.

HISTORY.

J. S. B., aged 24 years. Air gunner. First noticed serious trouble with vision in March, 1945. When using binocular reflector sights, during advanced training at an operational training unit, the target became double. He has also inclined his head to the left all his life, especially when reading.

Vision :—R. 6/6. L. 6/6.

Movements :—Defective elevation of right eye to left. Defective depression of left eye to left.

Diplopia :—Vertical upwards and downwards to the left.

Maddox rod :—Esophoria 5 Δ. L/R 3 Δ.

Synoptophore :—

R.	C.	L.	
FL +3° L/R 1 Δ	+4° L/R 5 Δ	+5° L/R 17 Δ +	} Varies.
FR +5° L/R 3 Δ	+5° L/R 6 Δ	+8° L/R 13 Δ +	
Fusion +4° 0, breaks L/R 5° to L. of midline.			

Adduction to +10°. Abduction to +2°.

Hess chart :—Paresis of left inferior rectus. Gross overaction of right superior oblique.

Diagnosis :—Paresis of left inferior rectus.

OPERATION. June 30, 1945.

Recession left superior rectus 3 mm.

POST-OPERATIVE MEASUREMENTS. July 5, 1945.

Synoptophore : R. C. L.
 Angle FL zero | 0° L/R 2 Δ incyclo. 5° | +3° L/R 22 Δ incyclo 5°.
 FR. L/R 1 Δ | 0° L/R 2 Δ-3 Δ incyclo. 2° | +7° L/R 17 Δ.

Fusion 0° ahead, holds to 15°-15°, then marked L/R.

Improved on laevo-elevation but diplopia on laevo version.

OPERATION. August 9, 1945.

Recession right superior oblique 3.5 mm.

POST-OPERATIVE MEASUREMENTS.

Ocular Movements :—Better to left, though still some L/R which is greater on elevation. To right there is defective depression of right eye and R/L : with incyclophoria increases on depression ? adhesions of right superior rectus limiting downward movement of the eye.

Synoptophore (August 17, 1945) :—R.—+8° R/L 8 Δ-10 Δ incyclo. 2° C. : +4° R L 2 Δ. L. : -8 L/R 14 Δ-15 Δ incyclo 5°.
 Fusion 0 0, centre breaking to right and left.

Fig. 6. Case No. 2.

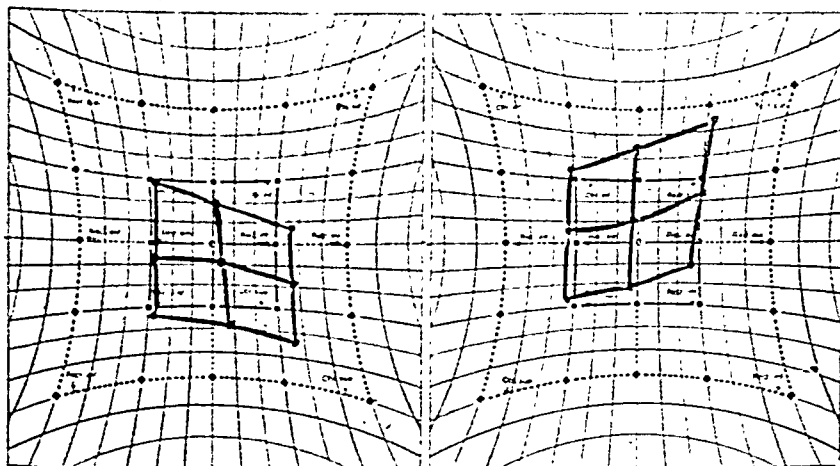
Orth. Date: 1.17.

Left Eye

Vertical n. of an. error
0.25 D.

Right Eye

Vertical n. of an. error
0.25 D.



Orth. Date: 1.17.

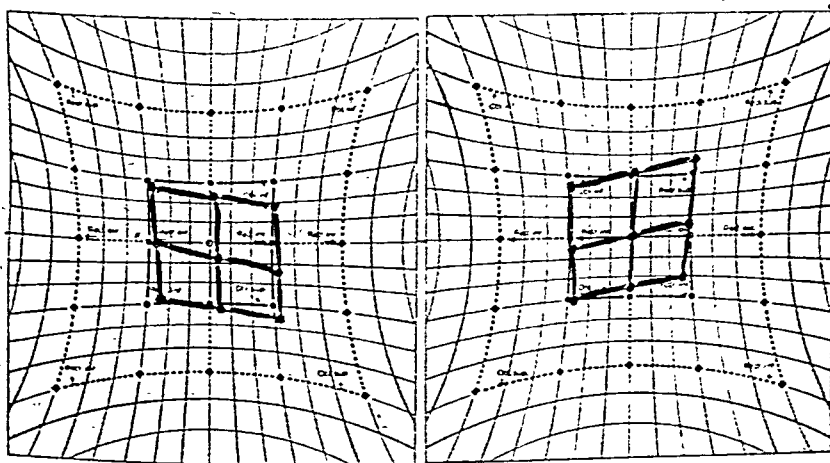
Orth. Date: 1.17.

Left Eye

Hyperopia 0.25 D.

Right Eye

Hyperopia 0.25 D.



OPERATION. August 20, 1945.
Removal of catgut stitches from right superior oblique and recession of right superior rectus.

POST-OPERATIVE MEASUREMENTS. August 22, 1945.
Synoptophore :—S.P.—R. : 0. C. : L/R 12 Δ -14 Δ . L. : L/R++.

Movements :—Defective elevation of right eye, to keep eye padded.
Synoptophore (August 30, 1945) :—
S.P. angle +7° L/R 4 Δ centre.
Fusion +4° 0, breaks 10° to left.
M.R. F.R. Eso. 7 Δ L/R 2 Δ .
F.L. Eso 4 Δ L/R 7 Δ .

Fourteen days sick leave.

OPERATION. September 24, 1945.
Tenotomy of left superior rectus.

POST-OPERATIVE MEASUREMENTS. November 3, 1945.
Ocular Movements :—Defective R. dextro-depression, with downshoot of L.E.
Diplopia :—In all fields, except for limited field of S.B.V. straight ahead.

Hyperphoria and cyclophoria increasing to R.
Maddox rod :—F.L. = Eso. 8 Δ R/L 3 Δ -4 Δ . F.R. = Eso. 7 Δ R/L 1 Δ .
Maddox wing :—Eso. 3° R/L 1°. Excycl. 3°.

Cover Test :—S.L.C.R.R. and R/L 7 Δ F.R.
Cyclophoria :—On dextro-version, R. Excycl. 4°.
Synoptophore :—S.M.P. angle—F.R. 20° R. = +5° R/L 5 Δ . Excycl. 4°.
0 = +5° R/L 3 Δ . Excycl. 2°.
20° L. = +5° 0.
F.L. 20° R. = +5° R/L 3. Excycl. 8.
0 = +5° R/L 1. Excycl. 5.
20° L. = +5°

Fusion +3° R/L 2 Δ . Excycl. 3°.
Can hold S.V. "buckets" at +3° R/L 2 Δ . 5° R. and 8° L.

Hyperphoria increasing to the R.
Adduction +20°. Abduction -1°.

2 Δ Prism base 45° R. 2 Δ Prism base 225° L. Ordered for constant wear,
pending further operation.

November 11, 1945. Adduction now +40° with increasing R. excyl.
The condition is still very unsteady. To proceed on leave for 2/12 and then
for re-admission. At present the most troublesome feature is the R. excyclophoria,
which worries him considerably. The hyperphoria is relieved to a certain extent
by the clip-on prisms, at eye-level.

March 17, 1946. Re-admitted to hospital. Eyes more settled. Measurements as
before.

Maddox rod :—Eso. 10 Δ R/L 5 Δ F/L. Eso. 10 Δ R/L 1 Δ F.R.
Maddox wing :—Eso. 5° R/L 2°. Excycl. 6°. 20° R. = +8° R/L 6 $\frac{1}{2}$ Δ . R. Excycl. 6°.
Synoptophore :—S.M.P. F.R. 20° R. = +6° R/L 3 $\frac{1}{2}$ Δ . R. Excycl. 4°.
0 = +6° R/L 1 Δ . No cyclo.
20° L. = 6° R/L 1 Δ . No cyclo.

F.L. +7° R/L 6 Δ . R. Excycl. 8°.
+6° R/L 3 Δ R. Excycl. 5°.
+6° 0. No cyclo.

Fusion +5° R/L 1 Δ . R. Excycl. 4°. R. Infraduction = 2 Δ .
Fusion held 8° R. and L.
Adduction +42°. Abduction to 0°.

Myectomy Right Inferior Oblique (March 19, 1946) :—Greatly improved. Much
larger binocular field.

Maddox rod :—Eso. 8 Δ 0 F.R. Eso. 8 Δ R/L 1 Δ F.L. +4° R/L 3 Δ .
Synoptophore :—F.R. 20° R. = +4° R/L 2 Δ . No cyclo. F.L. +4° 0
0° = +4° 0 No cyclo. +4° L/R 10 Δ .
20° L. = +4° L/R 8 Δ . No cyclo.

Fusion +2° 0 held 15° R. and 10° L.
Adduction +45°. Abduction -3°.

ON DISCHARGE FROM HOSPITAL.

Ocular movements improved. Still marked L. hyper. on laevo-version and eleva-
tion. Can read, etc., without discomfort. Single binocular field of 40° to 3/330.
Very satisfied with result, and can avoid residual diplopia by turning the head.
Prisms no longer necessary.

ANNOTATION

Through a Glass, Darkly

A note on contact lenses in a recent issue of the Journal has prompted a colleague who recently visited America to send a newspaper cutting which reads as follows:

"Anthony Aiello, convicted killer, was captured yesterday afternoon in Brooklyn in an anticlimatic last chapter in the round up of nine prisoners who broke out of the Raymond Street jail three months ago . . . he came back two weeks ago . . . got the rest of his cash and spent one hundred dollars for a pair of contact lenses to replace the identifying glasses."

This seems to bring right up to date the long list of favours which the science of optics has bestowed upon the malefactor, garnished with those bright, hard little touches of commercial efficiency which seem so characteristic of the American criminal. As yet the English criminal has not got as far as this; but then neither has the English contact lens industry; it is doubtful whether he could have arranged an appointment in this country in three months. We go about these matters more deliberately.

Nevertheless our criminals have not altogether despised the benefits which science has to offer. It was the operation of the laws of geometrical optics which darkened the lantern in the sturdy grasp of the unwitting Bill Sykes, in that tidier, simpler pre-Einstein era when the rays of light, like the British Army of the day, could be relied upon to manoeuvre in thin straight lines.

Perhaps this American note is the writing on the wall; perhaps spectacle-making and spectacle-wearing have passed the peak and must fall into decay and disuse. It is not easy to visualise, however, an ophthalmic world bereft of those twin diseases of vitreophobia and vitreophilia—the dread of glasses and the morbid craving for them—which are commonplaces of the consulting room. Will contact lenses solve the problem of the boy who throws away his spectacles the day after he leaves school or the young female presbyope who considers the occasional use of reading lenses a fate worse than death? We doubt it. But even these disorders pale before the iron determination of the glasses addict. From the voluminous handbag, or in the most intractable cases, from the capacious reticule, come pair after pair of spectacles, with lenses of every conceivable shape and hue, agreeing only in dioptric power; they have been prescribed by opticians, by ophthalmic surgeons, by half the great names of Harley Street, but all have been found wanting. Undeterred by the chastening power of experience, nothing will satisfy the craving but bigger and better lenses, of

greater 'magnifying power', combined of course with a corresponding reduction in the transmission of the well known baleful properties of light.

Not even contact lenses can solve this problem; 'not the labours of my hands, can fulfil thy law's demands'. There is perhaps hope for the refractionists yet, if only for the psychological comfort of their ministrations. It is difficult to see how contact lenses can meet the needs, the urgent imperative needs, of the young women (or the not so young women) who find the daylight too strong for them. Who is not familiar with the cold azure gaze of these maidens, entrenched behind the deepening tints of Crookes glass? With them the spectacle maker may take heart again; No contact lenses will fill their needs, for they are symbolic; no matter how carefully the conscious mind is dressing the shop window, with them the subconscious is already putting up the shutters.

FACULTY OF OPHTHALMOLOGISTS

In an effort to meet the objections of some Members and Associates that the activities of the Faculty were insufficiently advertised, and were only susceptible to comment once a year on the occasion of the Annual General Meeting, the Council at their last meeting on July 11, decided that an account of the more important actions and decisions of the Council at meetings should be prepared by the Honorary Secretary and submitted for publication to the Medical Press. An account of the last meeting, held on July 11 from 2.15 p.m. till 5 p.m. is appended.

Owing to the removal of Mr. P. M. Wood from Scotland to Halifax a vacancy had occurred among the Full-time Associate Representatives on the Council. It was decided to fill this casual vacancy by asking the Full-time Associates in Region 6 to vote for a representative. Only three voting papers had been returned and only one of them bore a vote. It was therefore decided to leave this Region unrepresented.

The President reported that, as "the appointed day" for the beginning of the National Health Service had been postponed, he had agreed with the British Medical Association's suggestion that the appointment of the medical members on the Professional Committees to prepare lists of participants in the Supplementary Eye Service should be deferred until the reaction of the general body of ophthalmologists to the Interim Report of the Eye Services Committee had been obtained. This was agreed.

A Hospital Services Committee has been set up with the following terms of reference:—

“To consider facilities for ophthalmological services, including education and research, under the National Health Service.”

Representatives from the Universities, Teaching and other Hospitals have been invited to form regional Sub-Committees in Scotland, the Provinces and London, and to submit plans for the consideration of the main Committee.

In connection with the question concerning the fee of four guineas per session for Local Authority Work, it was stated that the matter had been raised at the last B.M.A. Council and that it was not possible to alter the four guinea rate, as this had been agreed for all consultant and specialist work. but that the question of £2 17s. 6d. for refraction work being increased was still under consideration.

It was reported that the question of study visits to clinics abroad was in hand, and that it was likely that some would be arranged for next year.

It was learned in connection with the Joint Committee of the Faculty and the British Optical Association, which had been appointed to consider the training of contact lens fitters, that the Association of Dispensing Opticians had been invited to nominate a representative to serve on the Contact Lens Board, which is now in process of formation.

The suggestion that the Faculty should organise a discussion on the Education of Blind and Partially Sighted Children was referred to the Ophthalmological Section of the Royal Society of Medicine.

The Honorary Treasurer reported that he had collected £64 in private donations from members of the Council to assist an orthoptist who had suffered bilateral detachment of the retina, and had passed the money over.

The rest of the meeting was occupied by routine and domestic matters.

FRANK W. LAW,
Hon. Sec.

CORRESPONDENCE

COLOUR VISION IN THE CONSULTING ROOM

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIR,—Dr. Neubert's explanations in his letter in your August number are not convincing. In my letter I suggested that

the difference between the usually accepted 7·8 per cent. incidence of defective colour vision and the figure of 5·5 per cent. found by Dr. Neubert in his series, might be due to the methods of testing. It is no answer to this criticism to say that the difference must be due to the non-random method of sampling—it may be, but it might equally be the method of testing.

It is never a waste of space to describe accurately the methods of testing employed—in fact it is fundamental. No two people use the Ishihara plates in exactly the same way. Further, it is not sufficient to describe a multi-light lantern as having a rheostat, apertures of a certain size and filters approximating to those recommended by the International Committee for Aerial Investigation. In view of repeated rheostat adjustments it was pure chance if any two of the subjects examined on the multi-light lantern were tested under the same conditions, and in this type of work the colour temperature of the source cannot be ignored completely.

Surely the choice of title of an article does not remove the necessity for laboratory control of the tests employed. Without such control there is no justification for saying that the results presented show a different function for single as compared with multi-light lanterns. It is equally permissible to come to the same conclusion, without any experimental work whatever, when seated in an arm-chair by the fire.

I apologise for a second letter but I feel very strongly that the idea should not be spread abroad that the testing of colour vision and the classification of colour defects can be carried out cavalier-fashion with black-out shades and coffee-tins—unless lanterns made from such materials are properly standardised and used under clearly defined conditions. The efficient testing of colour vision is no easy task.

Yours sincerely,

JOHN GRIEVE.

MEDICAL SCHOOL, DUNDEE.

August 20, 1947.

NOTES

University of Glasgow
Department of
Ophthalmology
Autumn, 1947

DURING September and October a series of meetings will be held in the Department on Wednesdays at 8 p.m. The general arrangements will be similar to the series held last year. Tea will be served after the paper and a discussion will follow.

The meetings will be open to all medical practitioners and senior students interested in Ophthalmology.

September 24, Mr. F. Ridley, "Contact Lenses"; October 1, Dr. J. D. Fraser, "Industrial Cataract"; October 8, Prof. A. Loewenstein, "Pathology and Clinical Aspects of Ocular Glass-membranes"; October 15, Prof. A. J. Ballantyne, "Some problems in Ophthalmoscopic Diagnosis"; October 22, "Prof. W. J. B. Riddell, "Ophthalmology in Poland."

* * * *

National Society
for the
Prevention of Blindness

THE National Society for the Prevention of Blindness announces that it will hold a three-day conference, April, 5, 6 and 7, 1948, at the Hotel Radisson, Minneapolis, Minn. This conference will be of interest to persons who are directly or indirectly concerned with eye health and safety. Details concerning the programme may be obtained by writing directly to the Society at 1790, Broadway, New York 19, N.Y.

* * * *

The Estelle Doheny
Eye Foundation.
Eye Laboratory at
Los Angeles

THE Estelle Doheny Eye Foundation announces the establishment of an Eye Laboratory located at St. Vincent's Hospital in Los Angeles, designed to provide certain

modern ophthalmic facilities badly needed in Southern California.

The immediate functions of the laboratory are:—

1. To serve as a pathological laboratory for the diagnosis and registration of pathological specimens, with preparation of gross specimens and microscopic slides for ophthalmologists submitting specimens, and for the building up of a museum of eye pathology.

2. To serve as a bacteriological laboratory wherein diagnostic scrapings, smears and cultures can be studied, animal inoculations made, and the sensitivity of organisms to various drugs and antibiotics determined.

3. To provide facilities for fundus, gross, and slit-lamp photography, and to maintain a library of photographs and motion pictures for teaching of ophthalmology.

4. To provide an "Eye Bank" for Southern California, with registry of potential donors and recipients. Donor material will be collected, examined and distributed, and facilities may later be provided for limited instruction in corneal transplant procedures.

5. To distribute and loan certain drugs and equipment that are not otherwise available in the community.

6. To make available certain equipment for radiation therapy of the eye for use outside the laboratory.

Dr. Alan Woods, Professor of Ophthalmology at Johns Hopkins Medical School, Dr. Cecil O'Brien, Professor of Ophthalmology at

University of Iowa Medical School, and Dr. Phillips Thygeson, formerly Professor of Ophthalmology at Columbia University, and now Associate Professor at the University of California, will serve on the Advisory Board. Dr. A. Ray Irvine, Professor of Ophthalmology at the University of Southern California, will act as Chairman of the original board. It is contemplated that the Advisory Board will also include prominent business and professional leaders as well as representatives from the eye departments of local hospitals, medical schools, and from the Eye Section of the Los Angeles County Medical Association.

Dr. Peter Soudakoff, formerly Associate Professor of Ophthalmology at the Peking Union Medical College, will serve as full time pathologist at the laboratory. As part of the residency programme of the Eye Service of the Los Angeles County General Hospital and of certain local Veteran's Hospitals, each resident will spend two or three months at the laboratory. It is anticipated also that a research associate, granted a fellowship in basic ophthalmic research by the foundation, will be added to the laboratory staff from time to time.

In creating this perpetual charitable foundation dedicated to the conservation and restoration of eyesight, Mrs. Edward Doheny, with great generosity and discerning wisdom, has been careful to insure a flexibility of organization to take care of the immediate practical needs and at the same time provide for eventual development of much needed research in ophthalmology.

* * * *

Let there be Light The 32nd annual report of the National Society for the Prevention of Blindness, 1946 has the above title. The year has shown a resurgence of activities and the report indicates that the society is in a flourishing condition. During the year a three-day conference was held which was largely attended, the whole being most successful. During the year the Society has established nine scholarships of \$1000 for the education of qualified students interested in preparing for positions in the field of sight conservation and prevention of blindness. An industrial eye conservation programme has been worked out; and during a fortnight in June, the Society gave an intensive course of lectures and demonstrations at headquarters. Research is being actively undertaken, and nation-wide meetings are being organised. The campaign against glaucoma goes on. A summary of the year's finances completes the report.

* * * *

Corrigenda In the review of Kestenbaum's "Clinical Method of Neuro-Ophthalmologic Examination" p. 508, the publishers name should read Grune & Stratton, Inc. not Green & Stratton. And by an error the date 1949 was given for Troncoso's Treatise in gonioscopy in place of 1947.

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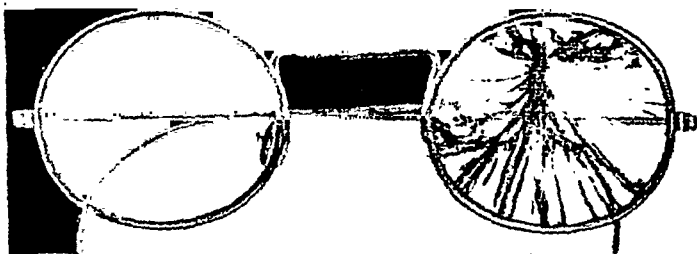
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THE BRITISH JOURNAL OF OPHTHALMOLOGY

NOVEMBER, 1947

COMMUNICATIONS

A CONSIDERATION OF ANIRIDIA, WITH A PEDIGREE

BY

P. H. BEATTIE

NORWICH

Introduction

Two or three patients in the family which is the subject of this enquiry had been treated in Addenbrooke's Hospital, Cambridge. On enquiry it was found that the defect was common in various related sibships, and although the affected people were living in villages scattered through the fen country between Cambridge and Peterborough, it was thought worth while to visit them all and record the condition of their eyes. In the future, members of such a family as this are less likely to remain domiciled in one locality, and an observer's task will be more difficult by virtue of the distances he will be obliged to cover to collect data from them all. The congenital abnormality shown in this particular family is a rare one. Few pedigrees of aniridia have been published in

recent years. Many which are widely quoted in authoritative textbooks are unreliable and have been constructed from second-hand reports. Many of the pedigrees collected by Julia Bell¹ are small and with little exact relevant detail. Julia Bell² has appealed for the suppression of the most remarkable aniridia pedigree published, that of Risley³, in which aniridia is reported in 111 out of 119 relatives but it was confirmed in the case of one individual only.

Strictly speaking the term "aniridia" is not accurate as some vestiges of iris have invariably been found in eyes which have been sectioned⁴, even though in life no trace was visible on ordinary clinical examination. The more limited term "irideremia" has been occasionally used but has not found general acceptance amongst ophthalmologists. For general purposes aniridia may be defined as "the entire absence of the iris or of a portion too great to justify the term coloboma."⁵ Any fragment which does exist is functionally useless.

All living affected persons in this family have been visited in their homes and examined there. In some cases they have been brought to hospital for more precise examination with slit-lamp, etc. Refractive errors were determined in their homes with an electric ophthalmoscope. Intra-ocular pressure was estimated digitally. Almost all non-affected descendants of II 1 were examined and a search made for any slight or intermediate change in the iris.

Thirty-one patients were found with bilateral abnormality of the iris. The abnormalities varied in gravity and have been subdivided thus—

Four cases with *coloboma of iris and/or hypoplasia of iris*.

Ten cases with aniridia either partial or complete of both eyes.

Twelve cases with bilateral aniridia and ectopia lentis.

One case with partial aniridia in R. eye and coloboma in L.

Four cases were not examined. Generations I and II are dead, as is Case III, 18. Careful questioning of their children and relatives make it certain that these people suffered from aniridia, but one cannot say whether or not the lenses were dislocated. Aniridia itself is such an obvious abnormality that lay testimony can probably be accepted, especially where the person involved was one of the family circle.

It is apparent from inspection of the pedigree that the three varieties of abnormality follow no special pattern of distribution. Cases with ectopia of lens are sometimes children of parents with uncomplicated aniridia, and vice versa. Those with *colobomata of iris* (or *hypoplasia of stroma*) occur in sibships affected with

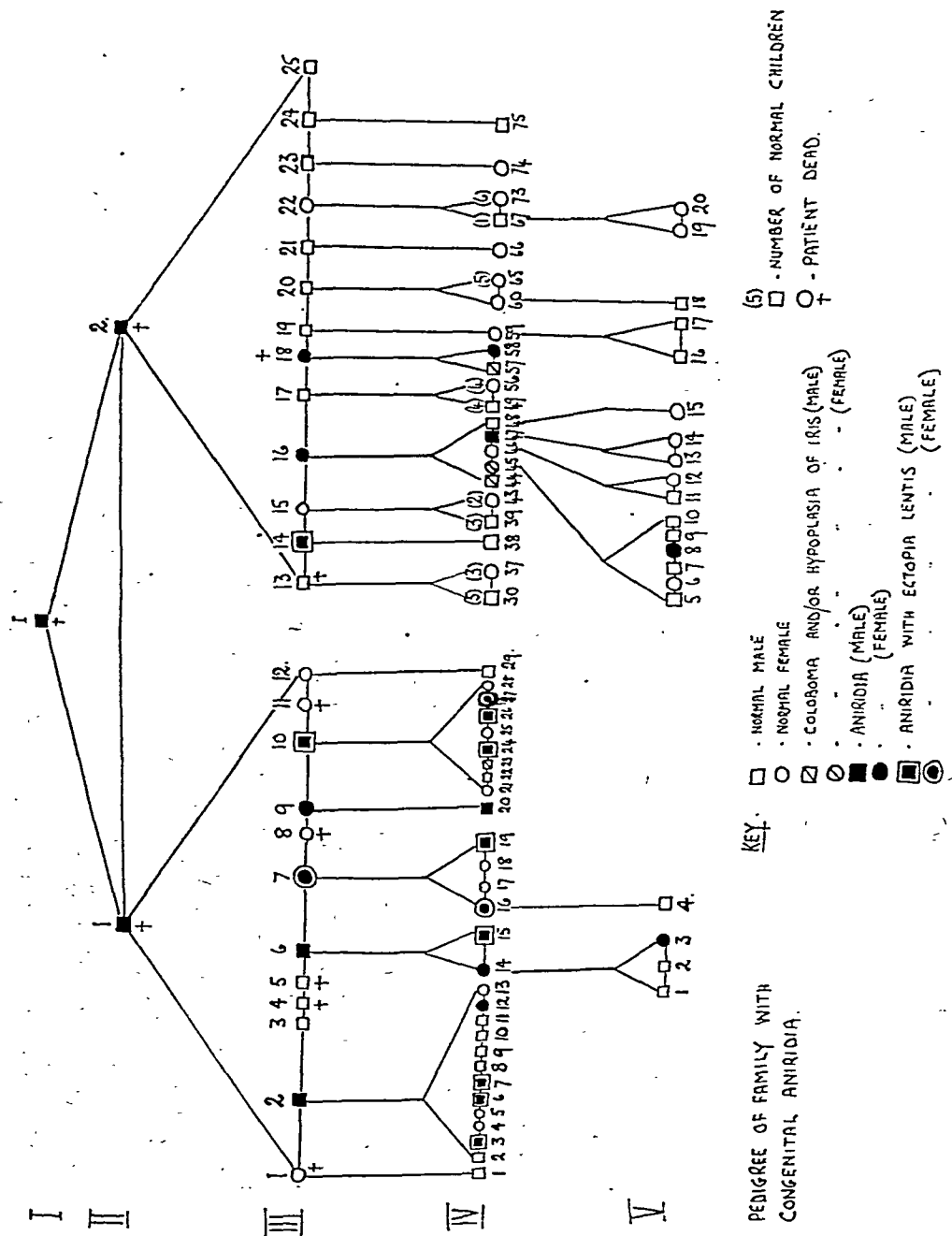
- p. 48. *Instead of "Secondary inhibitional paresis" after "3" read "Secondary underaction."*
Add "apparently" after "is" at end of 3rd line.
- p. 49. *Add "relatively" between "The" and "overacting" in 1st line.*
In 1st line of 5th para. change "overaction" to "contracture."
- p. 52. *Add to legends of Illustrations 23 and 24, "The broad lines, which are central, represent the red light."*
- p. 54. *Opposite p. 54, legend of Illustration 26, instead of "(3) then up and out" read "(3) than up and out."*
- p. 55. *Add to legend of Illustration 28, "The narrow lines, which are central, represent the green light."*
- p. 57. *In NOTE read "palsies of the inferior recti" instead of "R.I.R. palsy."*
- p. 61. *"Illustration 34" should be "Illustration 29."*
Section 1 refers to section 6 of Illustration 29.
Section 2 refers to section 2 of Illustration 29.
Section 5 refers to section 3 of Illustration 29.
Section 6 refers to section 4 of Illustration 29.
Section 7 refers to section 5 of Illustration 29.
- p. 63. *Omit sentence "Usually the inferior oblique is the affected muscle" in 3rd line of para. headed "(2) Primary or Congenital 'Overfunction'".*
- p. 65. *"Facial" should be "Fascial" in heading of section (c).*
- p. 67. *At end of 1st para. "Illustrations 29 & 30" should be "Illustrations 30 & 31."*
Under "Abduction in Elevation," "Diagram 32" should be "Illustration 33."
- p. 69. *At end of 2nd para. "Illustration 31" should be "Illustration 32."*
- p. 74. *In 2nd last para. "illustration No. 8" should be "Illustration No. 10."*
- p. 76. *Add "(a)" before "Unicocular Macular Projection."*
- p. 78. *"Illustration 33 (1)" should be "Illustrations 29 (1) & (2)."*
Add (b) before heading "The Division of Diplopia."
"Illustration 33 (2)" should be "Illustrations 29 (2) & (35)."
- p. 81. *"Illustration 33 (3)" should be "Illustration 29 (2)."*
In 2nd last line change "squares. A" to "squares; A, in Illustration 36."
- p. 82. *"—0°50'" should be "5°00'."*
- p. 83. *At the end of para. starting "Shirley L" read "contracture" instead of "overaction."*
- p. 89. *Add "ipsilateral" before "superior rectus" in Jackson's reference. The words "Bielschowsky (1945) condemned this operation" should follow.*

VERTICAL OCULAR DEVIATIONS

Addenda et Corrigenda for MONOGRAPH SUPPLEMENT NO. XII.

- p. 9. After "These" in 8th last line add "and the other influences concerned."
- p. 10. After "oblique muscles" in legend to Illustration 1 add "assisted by the vertical recti."
- p. 16. Add to legend of Illustration 3, "The narrow lines, which are central, represent the green light."
- p. 17. In 9th line "inferior rectus" should be "superior rectus" and be followed by "See Illustration 3."
- p. 18. Add "the" before "asymmetry" for "variation."
2nd para. Substitute "asymmetry" for "variation."
Last para. should precede 2nd para.
- p. 20. At end of para. marked (5) add "Illustration 5 (3) & (4)."
- p. 21. In 1st line omit "again" and after "is" add "another aspect of."
- p. 23. Replace last 2 sentences of 2nd para. by "This explanation is made more unlikely by the change to a downward rotation that occasionally occurs."
- p. 31. Omit last paragraph.
- p. 34. In 2nd last para. 8 lines from bottom of page, read "a contractive" instead of "an overaction."
- p. 35. In para. (3) change "up" to "vertically."
- p. 36. In 5th last line change "makes" to "may make."
- p. 37. In 1st line of legend add "muscle" after "oblique."
- p. 41. In 2nd line change "the roots" to "their roots."
- p. 42. In 14th line change "latter" to "later."
In 2nd last line of 4th para. add "a" after "due to."
2nd last para. should follow 1st sentence of 3rd last para. The next para. should be headed "DISCUSSION."
In last para. after "Chavasse" add "following Maddox and Peters."
- p. 47. 2nd column should read

- Sup. Rectus		
R. & L. R. L.		
1	2	4
—	6	8
1	6	11
—	2	7
2	16	30
15-0		



aniridia alone or with the graver condition of aniridia and ectopia lentis. A parent with coloboma is capable of transmitting the defect in a severer form to her children—IV 45 and V 8. The defects appear to occur in an unpredictable way amongst affected members. Unfortunately three of the colobomatous group have no children so that their genetic potentialities are not known.

Genetics

The defect is generally inherited from affected members of a family in a proportion of approximately 50 per cent. In this family 42 per cent. of the total descendants of II 1 are affected. Only 23 per cent. of the children of II 2 are affected, but the children of this 23 per cent. show an incidence of 62 per cent. Only affected members transmit the disease and normal children invariably have normal offspring. It therefore fulfils the conditions of a regular Mendelian dominant. In this family an affected person has invariably married a normal person and from this and the proportion of cases with aniridia we must assume the affected person is heterozygous—the mate in every case being homozygous for the recessive allelomorph. This may be illustrated by the following diagram⁶ :—

		One parent heterozygous affected	
One parent normal	Gametes	A	a
	a	Aa	aa
	a	Aa	aa
		i.e. 50% heterozygous affected 50% normal	

Being a dominant character the trait becomes apparent in a heterozygote. There are no cases in this pedigree, or indeed in the relevant literature, of two heterozygotes for the condition marrying each other and it is therefore impossible to say what appearance a homozygote for the condition would present. It is possible that lethal genes would accompany the homozygote condition, as in the case of the yellow mouse quoted by Corner⁷ and in numerous experiments with the *Drosophyla*. There is no accompanying infertility in aniridia as there is for example in neurofibromatosis⁸, nor in this pedigree is there evidence of mental or skeletal defects. We must assume that in some ancestor of I the

condition arose as a gene-mutation and thereafter, as in other alterations of the germ plasm, the defect was transmitted in accordance with mendelian principles as a regular dominant. Nothing is known of the cause of gene-mutations in human beings, or indeed of mammals generally, but in the insect world the normal production of mutations can be multiplied by means of thermal, X-ray stimulations of the germ plasm⁹. Plenty of records exist of aniridia arising without any known hereditary predisposition^{10, 11, 12} and in the case of a dominant of this type a new mutation must be postulated, especially where it is known that the defect is transmitted to approximately 50 per cent. of children¹³.

In recent years an attempt has been made to assess the mutation rate for various human abnormal conditions. J. B. S. Haldane¹⁴ began this work with a calculation of the mutation rate for haemophilia. Recently Mollenbach¹⁵ has estimated the mutation rate for aniridia, on the basis of his findings in Copenhagen, to be between 1:50,000 and 1:100,000.

The presence of three types of defect in one family without any apparent order in their incidence presents a difficult genetic problem. It is known that in hereditary defects a good deal of variability exists in different families and indeed in different sibships of the same family¹⁶. It may be that the iris form and structure are governed not by one but by many genes and that in the defects of varying gravity different numbers of genes are responsible. Waardenburg¹⁷ says "Since in the fruit fly *Drosophyla*—cytologically the most exhaustively studied animal—genes transmitting the colour of the eye or the appearance of the bristles have been found in every chromosome, it seems probable that the genes which determine normal structure and function of ocular tissues, and therefore those genes which determine their hereditary abnormalities, are distributed over several chromosomes." Environment can have no influence in a disorder where different primary defects occur in the same sibships and even between twins—IV 6 and 7. No consanguineous marriages are recorded in this pedigree. We must assume that "since there are families in which some have aniridia and others coloboma, whereas there are other families in which cases of coloboma occur in the absence of cases of aniridia, there must be distinct hereditary factors at work in the respective families, factors whose effects are quantitatively different."¹⁸ With a view to determining whether the abnormal gene (or genes) is linked with normal hereditary factors, blood and "taste-testing" examinations of several separate sibships were performed.

[A]

Case	III	2	—	A ₂
	IV	3		OIV
	IV	7		OIV
	IV	9		OIV
	IV	10		OIV

[B]

III	6	—	A ₂	pp.	M	rr
	Wife		O	P	MN	R _{2r}
IV	14	—	A ₂	pp.	M	rr
IV	15	—	A ₂	P	M	R _{2r}

[C]

III	7	—	A ₂	pp.	M	R ₁ R ₂
	Husband		O	P	MN	R _{1r}
IV	16		O	P	M	R ₁ R ₂
IV	17		(Not taken)			
IV	18		A ₂	P	MN	R ₁ R ₂
IV	19		O	P	M	R _{2r}

[D]

III	10	A ₂	pp.	M	R _{2r}
	Wife	B	P	MN	R _{1r}
IV	21	B	P	MN	R ₁ R ₂
IV	22	B	P	M	rr
IV	23	A ₂ B	P	M	R _{2r}
IV	24	A ₂ B	P	MN	R _{2r}
IV	25	B	pp.	MN	rr
IV	27	B	pp.	M	R ₁ R ₂

[E]

III	16	P	MN	R _{1r}
	Husband	P	M	R ₁ R ₁
IV	44	P	MN	R ₁ R ₁
IV	45	P	MN	R ₁ R ₁
IV	46	P	MN	R _{1r}
IV	47	P	M	R _{1r}
IV	48	P	MN	R ₁ R ₁

Note : Affected cases are underlined.

Taste-Testing with PHENYL-THIO-UREA.

A.	*III	6	—	Taster
	Wife		—	Non-Taster
	*IV	14	—	Taster
	*IV	15	—	Non-Taster

B.	*III	7	—	Taster
	Husband		—	Non-Taster
	*IV	16	—	Taster
	IV	17	—	Taster
	IV	18	—	Taster
	*IV	19	—	Taster

* Affected cases.

Taste-testing with PHENYL-THIO-UREA—*continued*

C. *III 10 — Taster	D. *III 16 — Non-Taster
Wife — Taster	Husband — Taster
All children — Tasters	*IV 44 — Taster
	*IV 45 — Taster
	IV 46 — Non-Taster
	*IV 47 — Non-Taster
	IV 48 — Not tested

* Affected cases.

These findings were scrutinized by Prof. R. A. Fisher, who thought there was slight statistical evidence of linkage to the ABO group. He considered greater numbers should be investigated and that the matter was worth pursuing. It was realized that even if linkage did exist the knowledge would be of no immediate practical value in a disability such as this which is present from birth (unlike Huntingdon's chorea where linkage with a known normal factor such as blood groups might enable a prediction to be made of those likely to develop the disease in later life¹⁹). Nevertheless it would be of some theoretical value as a contribution to the body of known linkages which must be enormously extended before substantial beginnings of a so-called chromosome map could be assembled for humans. At present a relatively complete map is a remote possibility, man being such a slow breeder and not available for controlled genetic experiments like the fruit fly *Drosophyla* from the intensive study of which so many facts have been learnt.

Embryology

No foetal eye affected with aniridia became available for study. It is obvious the disability is germinal: environment is not a factor of any importance, nor is it the result of any transplacental infection such as the foetal maldevelopments associated with maternal German measles²⁰. Many theories have been advanced to explain the mechanism of its production and no general agreement has been reached amongst investigators. Foetal eyes affected with aniridia are rarely examined.

The presence of an anterior polar cataract suggested to Treacher Collins²¹ that the defect was due to an abnormal and prolonged adhesion of the lens to the cornea (an analogous opacity is seen

after a perforated corneal ulcer). This would make it impossible for the iris to insinuate itself between these two structures. An anterior capsular cataract does occur in this pedigree, e.g., III 9, but it is not generally present—in fact in the youngest members the lenses and capsules are quite clear. One would also expect such an adhesion to be accompanied by an opacity in the substantia propria of the cornea, but such an opacity is not found in this series.

The mesodermal theory in Ida Mann's words²² suggests "the growth of the mesodermal iris is primarily abnormal and inhibits the normal forward growth of the ectoderm." Evidence has been adduced that colobomata of the iris are caused by the "abnormally long persistence of one, several or all the vessels which normally connect the circulus arteriosus iridis major with the terminal branches of the hyaloid vessel around the edge of the optic cup."²³ If all the vessels persist the growth of the iris would be impeded all the way round and the condition of aniridia result. The mesodermal iris which normally appears first would not perform the function of a scaffold along which the ectodermal elements could grow. Some support is lent to the mesodermal theory of genesis by cases IV 45, IV 44, IV 23; these have defects in the iris stroma only, with intact pigment layers beneath, suggesting at least that the defect is not due to ectodermal maldevelopment. These cases also have a marked proliferation of the pigmentary layers at the defective parts of the pupillary region, as though vigorous ectodermal tissue had attempted to close the mesodermal defect.

The ectodermal theory postulates that the rim of the optic cup which subsequently forms the neural layers of the iris does not grow forward at the proper time, that is at the 70-80 mm. stage²⁴. This theory would be more in line with the "organizer" hypothesis, whereby the optic cup is believed to have a "governing" function calling forth the lens²⁵. Evidence in its support is found in the ectodermal defects which accompany aniridia—absent fovea centralis, nystagmus, and anomalies of lens and retina. Nystagmus was found in 3 cases, choroido-retinal degeneration in 3 and (except for one case of nystagmus) all belonged to a sibship in which hereditary myopia was present—IV 2-13. In one case of nystagmus (III 9) a brisk foveal reflex could be seen on the R. side only, IV 13, a normal case in the same sibship had extensive peripheral pigmentation of the fundus in both eyes. The frequent incidence of ectopia lentis in this series may lend support to the ectodermal hypothesis, as this condition is thought to be due to a primary defect of the zonule, which results in a displacement of the lens when the ciliary ring opens out—the zonule being developed from tertiary vitreous and therefore an ectodermal structure²⁶.

Pathology

No first-hand facts can be given of the histology of the condition, as no eye in this family has become available for pathological examination. III 14 had the L. eye enucleated seven years ago, but no histological examination was made. The eye had been blind for many years and very red and painful for 5 months. Reports are not numerous in the literature, but all agree that a few tags at least of iris are found in every case of (clinically) complete aniridia²⁷. Treacher Collins²⁸ says "Microscopical examination of these cases seems to show that they are really predisposed to glaucoma, for not only do we find that the ciliary body terminates in a rudimentary iris, which if pushed forwards is quite sufficient to block the whole of the posterior surface of the ligamentum pectinatum, but that between this rudimentary iris and the lig. pect. there are abnormal adhesions."

Corneal opacities are stated to occur, but they were not seen in this series. Lenticular opacities were common but of no single type and supported no particular embryological theory. Opacities become more widespread and severe as affected patients grow older and ultimately cause incapacitating visual defects. It may be noted that generalized opacity of the lens occurs most early in two cases where they were dislocated, IV 3 and IV 6. Presumably the liability to cataract formation is due to impaired nutrition of the lens. The iris normally has some share in the interchange of fluids in the eye and its absence may be assumed to have a deleterious effect. If the ciliary processes are small or absent as they are sometimes said to be²⁹ this would further interfere with the fluid interchange. Possibly the absence of the constant contraction and dilatation of the iris may remove a normal stimulus and be responsible for a more sluggish circulation in the ciliary body, which would prevent a vigorous interchange of intra-ocular fluid.

The development of a generalized opacity may be accompanied by a swelling of the lens, with pressure on the iridial angle and the onset of secondary glaucoma—III 16. Why the presence of an ectopic lens greatly increases the tendency to glaucoma is not altogether clear. This complication does not appear to be especially common in families with congenital ectopia lentis without aniridia. It may be that the elongated fibres of the suspensory ligament permit very minute movements of the lens to take place, movements which are not always clinically visible, and these may set up a chronic irritation leading to glaucoma.

Some writers have found histological evidence of an absent fovea centralis³⁰, which is assumed to be the cause of the partial amblyopia and nystagmus frequently encountered in aniridia.

Clinical findings

A. CASES WITH COLOBOMA AND/OR HYPOPLASIA OF IRIS.

Four cases exhibiting small colobomata and/or hypoplasia of the iris. These patients suffer no disability. Visual acuity is normal, with no tendency to formation of lens opacities nor of raised intra-ocular pressure. They occur in sibships with normal and aniridic brothers and sisters and in each case are themselves children of one affected parent. From one case it is evident that they may transmit the disease in a more severe form to their children, *i.e.*, IV 45 who is the least abnormal of all affected members of her sibship, having slight hypoplasia of the superficial layers of the iris stroma revealing in patches the deeper pigmented layers, has transmitted the malady in a more severe form to one of her six children—V 8. The other five children are normal. IV 23 has a R. pupil of normal size, but the superficial layer of the iris has gaps shaped like the petals of a flower, exposing the deeper pigmented layers. The L. eye has a dilated and very feebly reacting pupil with a coloboma of the superficial stroma of the iris at 5 o'clock in the pupillary margin, but the gap is filled in with a proliferation of the deeper pigmented layers. IV 45 has a coloboma of the pupillary margin of the iris at 9 o'clock in R. eye, with numerous areas of hypoplasia of iris stroma. The L. eye has a pupillary margin coloboma at 5 o'clock, the gap being partially filled with deeper pigmented layers. There are also areas of hypoplasia in the lower and temporal regions of the iris, with a small complete breach in the iris at 5.30 o'clock, forming a second very small pupil. He sees 6/5 comfortably with each eye and reads J.1. IV 57 has a small pupillary coloboma of the iris in each eye downwards and outwards, with areas of superficial hypoplasia just above and below the colobomata. His vision is excellent; he was accepted as A1 for the army and passed his eye test for the railway.

This tendency for colobomata and hypoplasia of the iris to occur in aniridic families is frequently noted in the literature. Licsko³¹ noted atrophic anterior layers of iris in the child of an aniridic woman. Theobald³² records a woman with an atypical coloboma iridis in each eye having a child with aniridia and congenital squint. De Beck³³, Cross³⁴, Foster⁵, Polte³⁵ and others have recorded cases of intermingled aniridia and colobomata. In general it may be said that colobomata tend to occur in predominantly aniridic families, but Snell's family³⁶ exhibit the reverse finding—colobomata occurring in 5 generations with two individuals with aniridia. Complications are less frequent in

colobomatous eyes, but they do occur: *e.g.*, de Beck³³ records development of bilateral cataracts in a man aged 30 years, so disabling as to require extraction.

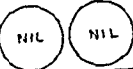


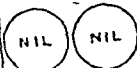




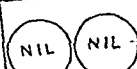

V 8 has a partial aniridia in the R. eye and a large coloboma of the iris of the L. eye directed downwards and slightly inwards. Such a case has been described by Waardenburg³⁷. In this child the lower margin of the lens could be clearly seen in the red reflex, even though there is no displacement of the lens—presumably because in a child of this age the lens is relatively small, whereas later on when the lens grows larger, the edge is obscured by the sclero-corneal junction. (The lens margin can be seen all the way round in case V 3, aged 2 years.)

B. CASES WITH SIMPLE ANIDIRIA. (Ten cases.)

The two predominant symptoms are photophobia and poor vision. Photophobia is a source of greater discomfort in the younger patients. V 3 and V 8 are obviously very uncomfortable in ordinary light. IV 12 wears tinted glasses only in very bright light. IV 20 served in the Middle East for 3 years without any special discomfort. The ophthalmoscope light caused lacrymation to IV 16 but normally she was quite comfortable. The older members of this group appeared to suffer no discomfort except III 2 who habitually wore tinted glasses. He, however, had had a bilateral lens extraction. Hamilton³⁸ suggested that lack of photophobia was due to abnormal insensitivity of the retinal elements. It may be, as this family shows that the retina is sensitive at first and gradually becomes more accustomed to excessive light as the patient grows older. Significant also is the fact that the lenses of aniridic patients develop scattered opacities as the patients grow older.

In none of these cases does vision reach normal standards. The following table gives the salient features of these ten cases.

From this it appears that vision deteriorates as the patient grows older. Case V 3 had transparent lenses but was too young for determination of vision. Case IV 12, aged 14, had 1 dioptré of myopic astigmatism, but correction of this did not improve visual acuity. IV 20 had an error of +2.50 sph. each eye, but this correction did not improve vision. IV 12 had clear lenses, and IV 20 a small localized opacity in L. which probably did not interfere with vision, yet both had mediocre vision. As the ages progressed the lenticular opacities increased, with exception of IV 28, who had slight dusty opacities only in the centre of each anterior capsule. III 2 was aphakic, both lenses having been removed when he was 34 for mature cataract. III 16 had a completely opaque lens in her R. eye, and a slightly less opacity

Case	IRIS PRESENT	Lens opacities*	Tension	Vision	Refractive error	Vision with correction
V 3 (age 2)		Clear	N	?	—	—
IV 12 (age 14)		Clear	N	6/36 6/24	-1.00D. cyl. at 180°	Not improved
IV 20 (age 21)		—	N	6/24 6/18	+2.50 sph.	Not improved
IV 14 (age 28)		—	N	6/24 6/18	?	—
IV 58 (age 30)		—	N	Not taken	+6.00 D. sphere	Vision good
IV 47 (age 32)		—	N	6/36 6/36	+6.00 D. sphere	6/12 6/12
III 9 (age 46)		—	N	6/18 6/60	+3.00 D. sphere	—
III 6 (age 50)		—	N	5/60 6/60	?	—
III 2 (age 57)		Aphakic-lens extractions 22 years ago	N	H.M. Certified blind	Now +8.00D. sph. eye is essentially myopic	—
III 16 (age 62)		—	+ ≠	Certified blind	?	—

(*) For details of lens opacities see case histories.

of L. III 7's vision has gradually declined: he was a soldier in the 1914-18 war. Thus it would appear that in the absence of lenticular changes vision is sub-normal from the beginning and that the opacities which accompany advancing years cause further gradual decline. Precisely why vision is poor in the absence of lenticular changes is not known. Some have produced histological evidence³⁹ that the macular area is abnormal, but no specimen from this series became available for pathological examination. A brisk foveal reflex was seen in a number of cases (see case histories). It has been pointed out by Alger⁴⁰ that the refraction of light both outside and inside the equator of the lens without the

iris diaphragm would cause a poor image and this lack of precision may deprive the macular area of effective stimulation in the early months of life when normal differentiation takes place.

Only in one case, III 16, was the presence of raised tension noted. The tension of the R. eye was higher than in L., but the rise was not marked. This is a small series, of course, and six of the cases are under 35 years of age. Nevertheless the findings are in striking contrast to those cases of aniridia complicated by ectopia lentis.

Although serious and incapacitating opacities of the lens in this small series have not developed until at any rate after the third decade, the literature records cases where a cataract showing "an expansion co-equal with the cornea" developed at 13⁴¹, and another patient at 15 or 16 years had both opaque lenses broken up by a needle⁴². Foster⁵ says that "Hirschberg watched a case from babyhood when the lens was clear and in place, until it was cataractous and shrunken and luxated at age of 10."

Glaucoma is generally accepted as a complication of aniridia. Foster⁵, reviewing the European and American literature at the end of the last century, found it present in 12 out of 164 cases. Julia Bell⁴³, however, says "it would appear that there is no marked liability to glaucoma in the aniridic patient." Treacher Collins^{44, 28, 45} repeatedly spoke of the greater predisposition of aniridic eyes to glaucoma, basing his opinion on histological evidence. Frost records a case in which one drop of homatropine precipitated an acute glaucoma (quoted in 38). In this family those with uncomplicated aniridia mostly have a hypermetropic error. From a survey of the literature this appears to be a common type of error^{46, 44}. This fact may account for the convergent squint present in V 8, and frequently noted by other observers in aniridia. De Beck³³, examining a lens after extraction from an aniridic patient, noted that its nucleus was excessively broad and thin. Patients suffering from traumatic aniridia are said to suffer from no defect of accommodative power⁴¹, but in this series it was noted that IV 58 with an error of +6.00 D. though only thirty years old, saw more satisfactorily with a second more powerful pair of glasses for close work, as did IV 47. IV 12 belongs to a sibship affected with myopia. This defect is not uncommon in aniridia families. Blair and Potter⁴⁷ have recorded two aniridic children of a colobomatous father, each child with high myopia and astigmatism. Foster⁵ records it as a common accompaniment of aniridia.

C. CASES WITH ANIRIDIA AND ECTOPIA LENTIS. (12 cases.)













In this family twelve patients have the additional primary complication of ectopic lenses as well as aniridia. The principal

features are briefly summarized in Table E. As in aniridia alone, photophobia is troublesome early in life. It is, for example, marked in IV 27 and her two brothers, IV 26 and IV 24, but in older patients the symptom is not complained of. Whether this is due to an acquired insensitivity of the retinal elements or to the development of corneal opacities is not easily decided. In general visual acuity is poorer than in uncomplicated aniridia, especially in such cases as IV 15, IV 19, IV 24, where the lower edge of the lens traverses the central portion of the large pupil and further interferes with the refractive system of the eye.

In each case the patient preferred, where he had the choice, to look through the phakic part of the eye, except III 7 who was helped by a +11.00 D. sphere correction in R. eye, even though the lens was not dislocated upwards as far as the centre of the cornea. The first three cases have a mixed astigmatism, myopia and hypermetropia "with the rule." IV 24 and IV 26 were slightly helped by the appropriate correction. IV 3, IV 6, and IV 7 were not refracted but had gross signs of myopia in the fundus, with marked evidence of retino-choroidal degeneration. In this sibship myopia was inherited from an affected aniridic father, III 2. IV 16 had a moderate hypermetropia; correction did not improve her distant vision, but was a considerable help with near vision. III 10 had a hypermetropic astigmatism; correction improved his vision slightly.

Cases V 24, 26, 27 and III 10 were examined with the slit-lamp. Cases IV 24 and 26 and III 10 showed the elongated fibres of the suspensory ligament below, placed regularly with no gaps and with a sturdy vitreous behind. IV 27 showed irregular fibres with a gap in the vertical meridian and a grossly degenerate vitreous behind. In IV 15 the suspensory ligament could be clearly seen with a loupe. The mixed astigmatism noted in cases IV 24, 26 and 27 could be explained by the weaker pull of the suspensory ligament below compared with the fibres laterally. None of these cases showed a highly myopic retracted spherical lens correctable with a -14.00 D. sphere, indicating complete absence of traction by the suspensory ligament⁴⁸.

Only in the two youngest were the lenses quite clear. Opacities developed in the second decade, but there was no regular gradation of density as the patients grew older. Dense cataracts were present in IV 3 and 7, whereas III 10, who is much older, had milky opacities present in both nuclei and a suggestion of lamination in the rest of the lens. On making a fundus examination one was conscious of a slight distortion in some cases, probably due to tilting of the lens. In no case was a coloboma of the lens observed. Tremulous lenses were observed in cases IV 24 and IV 3. Rayner

Case	Displacement of lens	IRIS	Visual acuity		Tension	Refractive Error	Lens opacities*
IV 27 Age 7		None	R H.M.	L. 6/36	++ +	Mixed astigmatism	—
IV 26 Age 11		None	6/36 with 6/36	6/60 glasses 6/36	++ ++	Mixed astigmatism	—
IV 24 Age 15		None	6/24	6/36	+ ++	Mixed astigmatism	—
IV 15 Age 15		None	6/24	6/36	Normal	Not refracted	—
IV 19 Age 18			6/60	6/24	++ N ⁺	Not refracted	—
IV 7 Age 19		None	Blind		Normal	Myopia	—
IV 6 Age 21		None	Blind		++ +	Myopia	—
IV 3 Age 28		None	Blind		++ ++	Myopia	—
IV 16 Age 32		None	6/36	6/60	Normal	Hyper- metropia	—
III 10 Age 44			6/60 with glasses 6/36	6/24 6/18+	Normal	Hyper- metropic astigmatism	—
III 7 Age 49		None	1/60 with glasses 6/60	<6/60 H.M. <6/60 H.M.	Normal	Not refracted	—
III 14 Age 64			Blind		++ L. eye enucleated for glaucoma		—

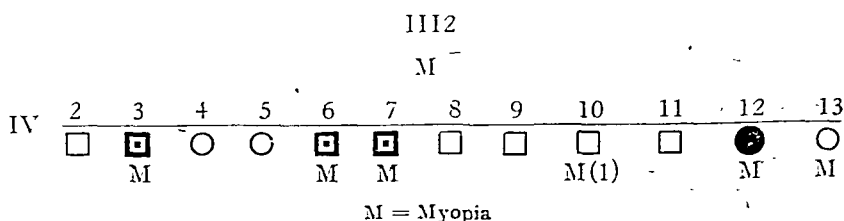
(*) For details see case histories

Batten⁴⁹ has recorded a case of bilateral aniridia and ectopia lentis: the R. lens was opaque and the L. partially so at 21 years old. The patient had never seen with the R. eye, but saw with the L. till she was 13 years old. Glaucoma had already developed.

The most striking difference on comparing these 12 patients with the ten having primary uncomplicated aniridia is in the incidence of glaucoma. Seven patients with ectopic lenses have glaucoma, whereas only one of the aniridia patients has slightly raised tension and she is the oldest of the group. Here IV 27, aged 7 has bilateral glaucoma, with an atrophic cupped disc observed, though in some eyes it was difficult to see the disc clearly or at all. Presumably the development of glaucoma is encouraged by the presence of ectopic lenses, but it is not clear why this should be so. Families showing ectopia lentis alone as a hereditary abnormality do not normally have a very high incidence of glaucoma. It will be observed that in five of the seven cases of glaucoma here there is no clinical evidence of any iris tissue present, and this, with possibly the particular formation of the iridial angle, may be a contributory cause of the raised tension.

Miscellaneous findings

The ocular defect is not accompanied by any skeletal abnormality, as, for instance, arachnodactyly, which is sometimes associated with congenital ectopia lentis. There are, however, some miscellaneous defects in the family which may be noted here. IV 7 is excessively fat, with a lazy good-humoured temperament suggestive of a pituitary dysfunction. X-ray examination of the skull revealed "a sella of physiologically small type." His twin brother, IV 8, is normal in all respects. Whilst hypermetropia is the predominating refractive error in this family, one sibship is found in which myopia is inherited from an affected father.



III 2, IV 3, IV 6 and IV 7 are all certified blind. IV 10 has myopia in R. eye only. IV 2 has a small myopia and astigmatic error (with aniridia). IV 13 has about 3 dioptres of myopia in

each eye. Stephenson¹⁰ records myopia of 6.00 D. in two members of an aniridia family, but in general hypermetropia appears to be more common. Blue sclerotics are found in one sibship—IV 24, IV 26 and IV 27. In IV 24 the sclerotics are duck-egg colour, but a deeper shade of blue is present in cases IV 26 and IV 27. In all three cases the anomaly is associated with aniridia, ectopia lentis and secondary glaucoma. Blue sclerotics are recorded in an aniridic family by Mohr⁵⁰.

Mental defect associated with aniridia is reported by Velhagen⁵¹, L. Polte³⁵, Stephenson¹⁰. No cases of subnormal intelligence were encountered in this family, either amongst normal or affected members. A particularly robust sense of humour is noteworthy in many members.

Nystagmus is reputed to be 'a common finding in cases of aniridia^{52, 53}. Here it was met with three times only—in cases IV 3 and IV 7 and III 9. Cases IV 3 and IV 7 are both certified blind, as stated above in connection with myopia. Case III 9 has a vertical nystagmus, the oscillations being so fine that they are discerned only on ophthalmoscopic examination. In the R. eye, which has 6/18 vision, a brisk foveal reflex can be observed. Vertical nystagmus was observed by J. B. Lewis⁴⁶ in one case of an aniridic family.

IV 24 has corneae measuring 9 mm. in diameter transversely, with slight ptosis and defective dental enamel. H. Page⁵³ mentions micro-cornea and microphthalmos in an aniridic family, as does A. H. Benson⁵⁴, whilst Hamilton³⁸ and Stephenson¹⁰ record defective dental enamel.

Two sibships were examined for colour vision and found to be normal. A curious inability correctly to pronounce the sibilant "S" was noted in the one sibship. The phrase "seen inside" becomes "shleen inshlide."

II

2

						L		L		L	L	L	
III	13	14	15	16	17	18	19	20	21	22	23	24	25

L = Lispings

None of the children of affected members has this lisp, nor from enquiries made does it appear to have been present in the case of II 2 or his wife. The mouths were not examined for any abnormal attachments of the tongue.

Treatment

(a) Cases with colobomata and/or hypoplasia require no treatment.

(b) In cases with aniridia the photophobia, in this family at least, appears to be adequately relieved by dark glasses. McKie Reid⁵⁵ has recorded a case where an adult with aniridia was relieved of his discomfort in bright light by wearing contact lenses with an artificial iris and a central pupil of normal size. His vision was improved to 6/9 by this treatment and troublesome photophobia abolished. This method may be applicable to adults, but in this series it is the children who complain mostly of discomfort. Alger⁴⁰ suggests tattooing of the cornea as being effective in cutting out peripheral light rays thus abolishing photophobia and maintaining an effective stimulus of the macula, which may be necessary for its normal development. He records a marked improvement in a young girl whom he treated in this way. This method might be satisfactory in young children if a satisfactory technique of tattooing were employed.

For the glaucoma which develops in aniridia trephining may be tried, but there is little reason to suppose it is effective in reducing tension. Wiener and Alvis⁵⁶ bluntly say operative treatment is hopeless. In this series IV 12 had a prophylactic trephine at seven years of age (she is now aged 14 years), but no filtration bleb is visible. She has retained a normal tension, but so have other untreated cases. Hudson⁵⁷ records the case of a baby aged 7 weeks with aniridia and bilateral acute glaucoma, which was relieved by a paracentesis and subsequent trephine operations. Satisfactory intra-ocular pressure was established, but there is no record of the case being followed up. III 14, the only one in the uncomplicated aniridia cases with raised tension, has bilateral cataracts: it may be the development of cataract has been accompanied by some swelling of the lens which has helped to block the iridial angle with rudimentary iris tags.

Eyes with aniridia have an undoubted predisposition to early lens changes, as this series abundantly shows. If the opacities proceed to cause serious impairment of vision, ordinary extraction of the mature lens does not appear to be accompanied by serious technical difficulty or risk. III 2 had a mature lens removed from each eye without any complication and went back later for capsulotomy. There is no report in the literature of a series of extractions in aniridia. De Beck³³ gives an account of extraction of lenses in two brothers, one with aniridia and the other with bilateral colobomata. Vitreous was lost and cyclitis occurred in each eye, but ultimate vision was good in one eye in each patient.

—in each case the eye in which cataract had developed more recently, which led De Beck to suppose that undue delay in operating on an aniridic cataract might make the prognosis poorer. Foster⁵ wrote that "a number of operators have testified that a greater degree of cyclitis or other destructive inflammation attends their removal than is usually the case." This may possibly be due to the aniridia eye being relatively less vascular in the anterior segment, having no iris framework for vessels, and in consequence, less able to absorb foreign protein from the lens and overcome any slight infection. The risk of complications should not deter an operator from attempting to remove an opaque lens. The patient has nothing to lose. Possibly an intracapsular extraction would be less likely to be attended by subsequent inflammation as there would then be no irritating lens protein left in the anterior chamber. Treacher Collins⁴¹ records an extraction where the patient regained vision 6/24 with +16 D., and J.4 with +20 D. In younger patients the cataract may be broken up by a needle and will sometimes rapidly absorb⁴², but a rise of tension may occur necessitating repeated paracentesis⁵⁸.

(c) *Aniridia with ectopia lentis*.—As in uncomplicated aniridia, photophobia may be relieved by dark glasses. Tattooing the peripheral areas of the cornea would obscure the lens and make estimation of its later condition difficult. In this series an attempt was made to improve vision by correcting the refractive error. Where possible to make an accurate estimation, refraction through the lens was found to be a mixed astigmatism, due presumably to the unequal pull of the zonule fibres. Correction in a few instances, e.g., III 10 and IV 24, made a little improvement. In no case was the lens found to be highly myopic, which would be expected if the suspensory ligament were defective in its whole circumference, a condition sometimes found in familial ectopia lentis and congenital microphakia. In one case only, III 7, was it possible to improve vision by an aphakic correction, and this in spite of the fact that the lens came well down in the pupil.

It is difficult to escape the conclusion that it is the ectopia lentis in combination with aniridia which is responsible for the high percentage of glaucoma in this series. To prevent the development of glaucoma by an early attack upon the lens would therefore appear to be the most rational procedure. Development of opacities in the lens are not necessary to produce glaucoma. The presence of an atrophic and cupped disc on R. side and raised tension on L. in case IV 27 at the age of seven years suggests that treatment at the earliest possible age is indicated. As removal of the lens would be a very hazardous procedure, almost certain to be accompanied

by vitreous loss on account of the difficulty of getting behind an upwardly dislocated lens, the first measure should be a needling of the lens in the hope of rapid absorption. If this were successful the eye would then be in the position of aniridia only with its more favourable prognosis, and the awkward optical disabilities caused by a dual refractive system would be removed. It would be possible to make a simple aphakic correction without the intervention of the lower border of the lens. If raised tension should develop after absorption of the lens, a trephine could be tried.

Case Histories of Aniridia Family

I 2. This man's eyes are known to have been abnormal.

II 1. According to his eldest son, this man's eyes were affected with the family complaint, but he was able to work as an agricultural labourer till well over 70. When very old he could see to fell trees and, in fact, was never obliged to retire because of poor sight.

II 2. J. W. This man's vision began to deteriorate when he was about 58 years old. He had looked after a pumping station in the Fen country satisfactorily, but during the last 20 years of his life his vision was very defective.

III 1. Deceased. Had normal eyes.

III 2. W. G. W., aged 57 years. This man's vision was never very good. It began to deteriorate seriously about 30 years ago. In 1922 he was admitted to the Royal Eye Hospital for a R. extraction and needling, and the L. eye was operated on in the following year. The lenses were opaque but there is no reference to ectopia lentis.

There is no clinical evidence of the iris. Both eyes are aphakic: some posterior capsule is visible in both eyes. Tension—normal both eyes. Refraction—+8.00 D. sph +2.00 D. cyl. at 180 deg. Both fundi show myopic crescents and some central retinal degeneration. L. eye has a marked divergent squint. Vision—hand movements only. Prefers to wear slightly tinted glasses.

III 3. Normal eyes and vision.

III 4. III 5. Both males which died in infancy. Their eyes were normal.

III 6. Mr. S. W., aged 50 years. This man was in the 1914-1918 war. He now works in a brickyard and rides a bicycle to his work. Does not wear glasses. He gets very slight photophobia in bright light.

R. eye:—Complete clinical aniridia present. Tension normal. Slight central anterior lens opacity present and a wedge-shaped opacity which appears to involve whole thickness of lens at 5 o'clock. Vision—5/60.

L. eye:—Very narrow strip of iris present on nasal side. Tension normal. Milky nuclear opacity of lens present and a wedge-shaped opacity at 5 o'clock, as in other eye. Vision 6/60.

Patient himself thinks his vision has not declined in recent years, but his brothers are quite sure it has. No abnormality seen in fundi, but foveal reflexes could not be seen on account of lenticular opacities.

III 7. Mrs. F. M. This woman is a housewife. She has brought up four children and still manages to do house work.

R. eye:—Complete clinical aniridia present, with upward dislocation of lens. Slight opacities present throughout lens. Fundus seen through aphakic area appears normal. Tension—normal. Can read J.10. Distant vision is 1/60, but with a +11.00 D. sphere vision improves to 6/60.

L. eye:—Marked divergent squint present. Complete clinical aniridia, with upward dislocation of lens. Moderately dense opacities present throughout lens. Fundus not seen through aphakic area on account of vitreous haze. Vision—hand movements only. Tension—normal.

III 8. Female baby which died in infancy: normal vision.

III 9. Mrs. H. W., aged 46 years. This woman, unlike her brothers and sisters, has worn glasses for many years. A very fine *vertical* nystagmus is present in both eyes.

R. eye :—A narrow fringe of blue iris is present round the whole circumference, with a gap between 4 and 6 o'clock. The iris has a dark pigmented fringe, which looks like proliferation of the ectodermal layers. A small central anterior polar cataract is present with a circle of less dense opacities on the anterior capsule, paracentral in position. Vision 6/18: wears about +3.00 D. spheres. Macular area seen on this side, with brisk foveal reflex.

L. eye :—A narrow fringe of iris, blue in colour, is present, except for a gap between 3 and 5 o'clock. A central anterior polar cataract present, with a paracentral ring of capsular opacities. Fundus normal, except that foveal reflex could not be observed. Vision 6/60.

III 10. Mr. S. W., aged 44 years. This man is not troubled in bright light. Beyond 5 or 6 yards away he cannot recognize anybody. Is an agricultural labourer.

R. eye :—Very narrow fringe of iris present on nasal side. Lens is dislocated upwards and slightly outwards. With slit-lamp regularly disposed fibres of the suspensory ligament could be clearly seen in the aphakic part of the eye. The lens showed a central nuclear milky opacity, with opacities surrounding this showing a tendency to lamination. Slight tremulousness of lens seen with slit-lamp. Tension—normal. Vision R. 6/60, improved to 6/36 with +1.50 D. cyl. at 170°.

L. eye :—Condition of iris and lens as described in *R. eye*. Lens slightly tremulous when viewed with slit-lamp. Tension—normal. Vision 6/24; with +0.75 D. sph. +1.0 D. cyl. 90° vision improves to 6/18+.

III 11. Female child which died in infancy. Eyes normal.

III 12. Mrs. P. M., aged 40 years. Slight hypermetropic error (+1.00 D. sphere), otherwise eyes are normal.

III 13. Mr. W. W., deceased. Eyes were normal.

III 14. Mr. E. W., aged 64. This man's vision has always been poor, but 10 or 11 years ago it became too bad for working. Prior to that he could work in the stockyard if cattle were brought to him, but he could not work in the fields.

R. eye :—A very narrow fringe of iris is present in the upper nasal quadrant, extending from 12 o'clock to 4 o'clock. The lens is ectopic, displaced directly upwards. Lens is opaque throughout, but less so in the upper nasal and upper temporal periphery. Lens is tremulous. A good red reflex is visible in aphakic part of eye, but no view of retina could be obtained as vitreous is hazy. Suspensory ligament could not be seen below. Tension—+. Vision—perception of light.

L. eye :—This was enucleated seven years ago following five of pain and redness in the eye. It had been blind for many years. Unfortunately, a pathological examination of the eye was not made.

III 15. Mrs. M. A. G. Normal eyes and normal vision.

III 16. Mrs. E. T. Patient is a housewife who brought up five children. She saw fairly well until about seven years ago, when her vision began to fail. Earlier in life she was tried with glasses, but these did not help her.

R. eye :—Partial aniridia present. A narrow band of blue iris extends from 9 o'clock to 5.30 o'clock, with a deeply pigmented inner fringe. The lens is uniformly opaque, having a mother-o'-pearl appearance. There is good projection of light in the eye. Tension—slightly raised.

L. eye :—Divergent squint present. Has never been able to see much with this eye. Partial aniridia present: the iris is slightly broader than on *R. side*, being completely absent between 6 o'clock and 3 o'clock. Paracentral opacities are present posteriorly in the lower temporal and nasal quadrants of the lens. Slight hazy opacity is present throughout the lens. The fundus can be faintly seen through the lens and appears normal. The appearance is that of a hypermetropic eye. Tension—slightly raised.

III 17. Normal eyes.

III 18. Mrs. A. R., deceased. This woman had abnormal eyes, but whether she had ectopia lentis is not known. She died of carcinoma at age of 54, but

for some years before her death vision had deteriorated very much. She had a lisp.

- III 19. Mr. F. W. Normal eyes.
- III 20. Mr. A. W. Normal eyes. Has a lisp.
- III 21. Mr. Z. W. Normal eyes.
- III 22. Mrs. F. L. Normal eyes. Has a lisp.
- III 23. Mr. L. L. Normal eyes. Has a lisp.
- III 24. Mr. H. W. Normal eyes. Has a lisp.
- III 25. Mr. S. W. Normal eyes.

IV 1. Has normal eyes.

IV 2. Mr. J. W. Has normal eyes and good vision. Was recently discharged from the Army and now works as a signaller on the railway.

IV 3. Mr. W. G. W. This man was certified as blind at age of nine years. Prior to that he had been able to see to go to school. He is now a basket-maker.

R. eye:—No iris visible. Lens is displaced upwards and outwards into the upper temporal quadrant of the cornea. It is uniformly opaque. A good view of the fundus is obtainable through aphakic area: the retina shows extensive central degeneration. Disc is very pale. Tension—+++. Vision—no perception of light.

L. eye:—No iris visible. The lower part of the displaced lens is present in the upper nasal quadrant of the "pupil" and is uniformly opaque. It is also tremulous. The fundus cannot be seen through the aphakic area on account of a very hazy vitreous. Tension—+++. No perception of light present.

IV 4. Miss M. P. W. Eyes normal. Vision 6/5:6/5.

IV 5. Miss V. W., aged 24 years. Eyes normal, apart from chronic blepharoconjunctivitis.

IV 6. Mr. K. W. This man works in a blind institute as a basket-maker.

R. eye:—Complete clinical aniridia. Lens is displaced upwards. There is a very circumscribed dense opacity, central in position, involving the capsular and subcapsular tissues. Lateral nystagmus present. R. disc is pale and atrophic and the whole fundus has appearance of a very myopic eye. Tension—+. Vision—approximately 1/60.

L. eye:—Complete clinical aniridia. Lens is displaced upwards and slightly nasalwards. Slight superficial central opacities of the lens. Vitreous is hazy on this side, but fundus appearance suggests greater myopia than on R. side. Tension + +. Vision—1/60. Nystagmus present.

IV 7. Mr. R. W., aged 19 years. This boy is certified blind. He works at home, working in a small timber business. He is very fat, rather lazy, but generally very good tempered. X-ray of the skull shows a "sella of physiologically small type." He is just able to discern light and darkness.

R. eye:—No iris visible. The lens is displaced upward and slightly towards temporal side: it is opaque and of pearly colour. Inferior to the lens, through the aphakic part of the eye, a dense mass of connective tissue strands can be seen in the vitreous on the temporal side. The fundus cannot be seen. Tension—normal.

L. eye:—No iris visible. The lens is displaced upwards, its lower border reaching about half way down the vertical diameter of the cornea. Diffuse opacities prevent ophthalmoscopic examination through the lens. The retina can be seen through the aphakic area: gross retino-choroidal degeneration is present in the central area, and the eye is obviously a very myopic one. Tension—normal.

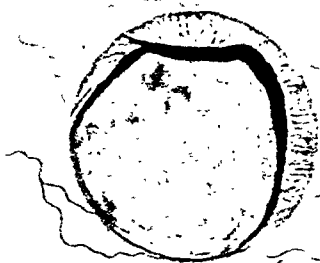
IV 8. C. W. This boy is a twin with IV 7, presumably a fraternal twin as there is little physical resemblance. Has a traumatic ptosis of L. eye, but otherwise his eyes are quite normal.

IV 9. P. W. This boy has just been discharged from the Marines. His eyes and vision are normal.

IV 10. F. W. No anatomical defect of eyes. L. eye is emmetropic, R. has 4 dioptries of myopia.

IV 11. C. W. Died at age of 5 months. Eyes were normal.

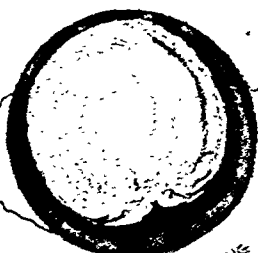
IV 12. G. W. This girl works as a hospital ward maid. Wears tinted glasses in very bright light. Bilateral trephine operation 7 years ago, but no filtration bleb can be seen.



III 16, Mrs. E.T.

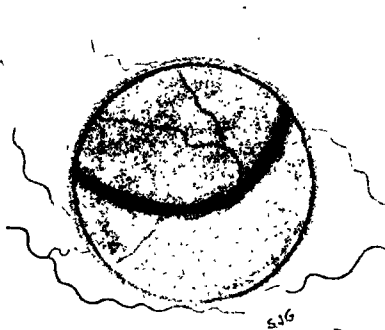
R. eye, focal illumination.

L. eye, red reflex.

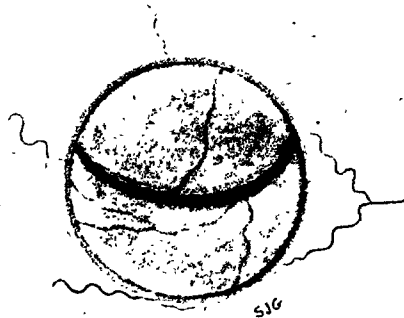


IV 7 (R.W.) R. eye, focal illumination.

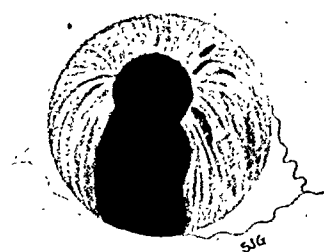
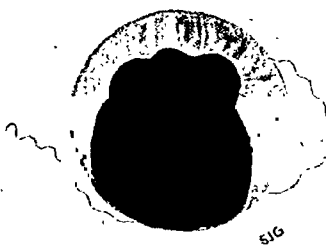
L. eye, red reflex.



IV 27. R. eye, red reflex.



L. eye, red reflex.



V 8. M.S., aged 4 years, focal illumination.

R. eye:—No iris visible. Lens in normal position and quite clear. Tension—normal. Vision—R. 6/36: small myopic and astigmatic error present, but vision is not improved by this correction.

L. eye:—No iris visible. Lens in normal position and clear. Tension—normal. Vision—L. 6/24. No improvement with small myopic and astigmatic correction. Both fundi normal, but poorly pigmented. Near vision with both eyes together is J.4.

IV 13. S. W., aged 13 years. This girl has 3.0 dioptries of myopia. Both retinae show numerous small pigmented spots in the periphery. A large arterial loop is present on L. optic disc, extending forwards into the vitreous.

IV 14. D. W. Says she has slight discomfort in bright light. Examination with ophthalmoscope causes some lachrymation.

R. eye:—No iris visible. Lens is in normal position. Scattered opacities are present on anterior capsule and in anterior cortex of lens, especially peripherally. Fundus normal; foveal reflex seen. Small hypermetropic error present. Tension—normal. Vision 6/24.

L. eye:—No iris visible. Lens clear and in normal position. Fundus normal: macular area normal, with brisk foveal reflex. Tension—normal. Small hypermetropic error present. Vision 6/18.

IV 15. Aged 15 years. Works on the land. Does not wear glasses. Cycles to his work.

R. eye:—No iris visible. Lens displaced upwards and slightly towards temporal side. Small grey opacities present in periphery of lens. View of fundus through the lens is distorted (? due to some tilting of the lens). Through the aphakic part the fundus appears to be normal: a good foveal reflex present. With a loupe the elongated fibres of the suspensory ligament can be clearly seen below the lens. Tension—normal. Vision—6/24.

L. eye:—No iris visible. Lens displaced upwards, with fibres of suspensory ligament clearly visible with loupe. Slight diffuse opacities present in lens. Disc visible through both lens and aphakic part. Foveal reflex not seen on L. Vision—6/36. Tension—normal. With both eyes can read J.4, holding print very close to eyes.

IV 16. Mrs. E. S., aged 32 years. Says she sees well, but is "a bit short-sighted." Not troubled by photophobia. She sees exceptionally well at night. Both corneal are small.

R. eye:—No iris visible. Lens displaced upwards very slightly. Very slight central anterior opacity of lens, with more marked paracentral opacity. Tension—normal. Vision—6/36. Reads J.12.

L. eye:—No iris, and lens dislocated as in R. eye. Slight paracentral opacity in lens anteriorly. Tension—normal. Reads J.14. Vision—6/60.

This woman has +3.00 dioptries hypermetropia in both eyes. Correction does not improve distant vision, but helps considerably for near work.

IV 17. I. M., aged 28 years. Normal eyes. Vision 6/6 : 6/6.

IV 18. I. M., aged 19 years. Normal eyes. Vision 6/6 : 6/6.

IV 19. Mr. O. M., aged 18 years. Works as farm labourer. Has very good physique.

R. eye:—Slight fringe of brown iris present above, extending from 9.30-3.30 o'clock, with a slight constriction at 1 o'clock. Lens is dislocated upward and slightly nasalwards, with lower edge of lens midway across the anterior chamber. Lens is slightly opaque throughout, with denser opaque dots in lower periphery. Vitreous slightly hazy. Fundus not clearly seen, but peripheral parts appear to be healthy. Tension—+ ±. Vision—6/60.

L. eye:—Slightly wider fringe of iris present, extending from 7.30-3.0 o'clock. Lens dislocated upwards and nasalwards. A wedge-shaped opacity present in posterior part of lens between 4-5 o'clock but lens otherwise clear. Vitreous clear. Tension—normal. Vision—6/24. Reads J.6.

IV 20. Mr. W. W., aged 22 years. This man served in the army in the Middle East. He was not particularly troubled by photophobia.

R. eye:—An extremely narrow fringe of blue iris is present on nasal side between 2.5 o'clock. Lens in normal position. Fundus—normal. Tension—normal. Vision—6/24. Good foveal reflex seen.

L. eye :—Very narrow fringe of iris present except between 2.30-5.0 o'clock where there is complete absence. At 9 o'clock and at 12 o'clock there is a proliferation of pigmentary (posterior) layers of iris. There is a small dense localized opacity on anterior capsule in lower temporal quadrant of lens. Fundus—normal. No foveal reflex seen. Tension—normal. Vision—6/18.

Both eyes have a hypermetropic error of +2.50, but this correction does not improve vision.

IV 21. Miss N. W. Normal eyes.

IV 22. B. W. Normal eyes.

IV 23. D. W.

R. eye :—The green stroma of iris has five gaps in it, shaped like petals of a flower, revealing the pigmented deeper layers. Pupil is of normal size. Fundus—normal. Vision—6/6 and J.2.

L. eye :—There is a partial (pupillary) coloboma of iris at 4.30 o'clock. The gap in the superficial stroma is filled in with proliferated pigment layers, so that pupillary red reflex is quite circular. Pupil reacts very feebly to light. Fundus—normal. Vision—6/6 and J.4.

IV 24. S. W., aged 15 years. Marked photophobia in bright light. Sclerotics are "duck-egg" blue shade. Corneae are small (9 mm. horizontal diameter), some ptosis present and palpebral fissure small. (An attempt was made to take a Schiötz reading, but there was insufficient room to accommodate the base of the instrument between the opened lids. Dental enamel is defective.

R. eye :—Complete clinical aniridia present. Lens dislocated upwards. Elongated fibres of suspensory ligament clearly seen below with slit-lamp. Lens has diffuse slight opacities, but of no special distribution. Lens slightly tremulous and appears to be tilted backwards above. Tension +. Mixed astigmatism present, but will accept only -3.00 D. cyl. at 165 deg., with which he sees 6/24. This is the same as without glasses, but definition is better. Good view of fundus through lens and aphakic portion of eye. Fundus—normal.

L. eye :—Aniridia complete. Position and condition of lens as for R. eye. Tension ++. Mixed astigmatism present, but accepts -2.00 D. cyl. at 180°, with which he sees 6/36. Good view of fundus not obtained because of slight vitreous haze. Lens slightly tremulous. Elongated fibres of suspensory ligament clearly seen.

IV 25. J. W., aged 14 years. Normal eyes.

IV 26. S. W., aged 11 years. Marked photophobia in bright light. Blue sclerotics present.

R. eye :—No iris present. Lens dislocated upwards. Lens clear but fundus difficult to see because of ? tilting of lens. Disc clearly seen through aphakic part of eye: fundus appears normal. Tension ++. Vision—6/36. Mixed astigmatism present. Sees J.14, holding print very closely to eye.

L. eye :—No iris present. Lens dislocated upwards as in R. eye, clear and slightly tilted. Aphakic portion clear, but disc difficult to see because of intervention of rim of lens. Tension ++. Vision—6/60, but improves to 6/36 with -2.50 D. cyl. at 180°. A mixed astigmatism is present, but the + constituent is not accepted. Vision is not improved on either side by correction of the aphakia. Sees J.14.

IV 27. J. W., aged 7 years. Schoolgirl.

R. eye :—Complete aniridia. Lens dislocated upwards. Lens and vitreous clear. An atrophic and cupped disc present, with no central vessels but all emerging from periphery of disc. Tension ++. Vision—6/60 and J.16, held very close. Mixed astigmatism present.

L. eye :—Complete aniridia. Lens clear, but dislocated upwards. L. fundus healthy. Tension +. Vision—6/36 and J.16. Vision not improved by correction (mixed astigmatism).

With the slit-lamp the elongated fibres of the suspensory ligament on each side could be seen very irregularly disposed and with a wedged shaped gap between 5.30 and 6.30 o'clock, suggestive of colobomata. The vitreous beyond was fluid and degenerate. A needling of R. lens was recently performed. This was followed by a lowering of intra-ocular tension, except for a transient rise lasting one day. The lens is now in process of absorption.

IV 28. W. W., aged 3 years. Normal eyes.

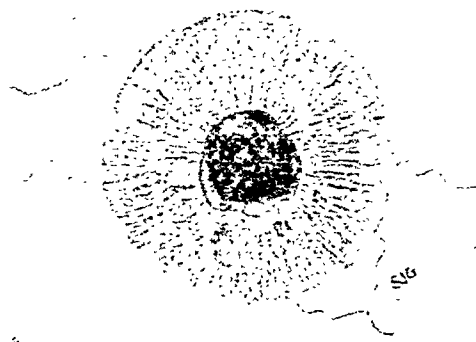
IV 29. Normal eyes.

IV 30-43. Normal eyes.

IV 44. G. W. T., aged 40 years. Is a carman. This man has no children, so that his genetic potentialities are not known.

R. eye:—Partial coloboma of iris present at 9 o'clock. Several areas of hypoplasia of stroma showing deeper pigmented layers in base of gaps. Some proliferation of posterior pigmented layers present in region of coloboma. One long strand of persistent pupillary membrane present. Lens and fundus normal. Vision—6/5.

L. eye:—Partial coloboma at 4.30 o'clock. Four small areas of hypoplasia of stroma, showing pigmented posterior layers of iris. One small area at 5.30 o'clock in which all layers of iris are absent and a good red reflex can be seen. Some



IV 45. Mrs. M. S. Left eye by focal illumination.

proliferation of posterior pigmented layers in region of coloboma. Lens and fundus—normal. Vision—6/6.

IV 45. Mrs. M. S., aged 38 years. This woman's eyes at a casual glance would pass for normal.

R. eye:—Normal except for a gap in the superficial stroma and proliferation of deeper pigmented layers at the pupillary margin, between 7-9 o'clock. Lens and fundus—normal. Vision—6/5.

L. eye:—A small area of hypoplasia of iris stroma at 6 o'clock, showing deeper pigmented layers. A few strands of stroma can be seen passing over the area. Three other smaller such areas are present in the lower temporal region. At the pupillary margin the stroma is deficient from 4-9 o'clock, with proliferation of posterior pigmentary layer. Lens and fundus—normal. Vision—6/12.

IV 46. Mrs. B. Normal eyes.

IV 47. Mr. J. T., aged 32 years. This man works in a sand-pit.

R. eye:—Cornea normal. A thin fringe of iris extends from 7.30 to 5 o'clock, with an area from 5 to 7.30 o'clock; completely without iris. The lens has a superficial paracentral opacity just below anterior capsule, and a deep opacity in the lower nasal quadrant. Fundi—normal. Tension—normal. Vision—6/36 with correction (+6.00 D. sphere) improves to 6/12.

L. eye:—Same distribution of iris as in the right eye. Deep lens opacity present in lower temporal quadrant. Fundus—normal. Tension—normal. Vision—6/36, with correction (+6.00 D. sphere) improves to 6/12.

There may be some impairment of accommodation present here, as he sees to read very much better with +7.00 D. The extra dioptré considerably impairs his distant vision.

IV 48. Mr. E. W. Normal eyes. Was a driver in the army.

IV 49-56. Normal eyes.

IV 57. Mr. W. R. This man is a lorry driver by trade. Was passed A1 for army and accepted for railway work.

R. eye:—Partial coloboma of iris down and out, with proliferation of pigmented posterior layers to fill the gap. Six small areas of localized hypoplasia of iris stroma. Lens and fundus—normal.

L. eye:—Partial coloboma of iris down and out, with same pigmentary proliferation as on R. side. Four areas of hypoplasia here in lower temporal quadrant. Lens and fundus—normal.

This man is not married and therefore his genetic potentialities are not known.

IV 58. Mrs. E. R., aged 30 years. Housewife. Sees well. Slight discomfort in bright light.

R. eye:—Small fringe of pale blue iris present extending from 10.30 to 4 o'clock, with notch at 1 o'clock. Lens not dislocated. A very faint central capsular opacity, with several flecks of thicker opacity present in inferior quadrants. Tension—normal. Fundus shows some small patches of old choroiditis in upper temporal quadrant. The anterior capsule of lens is flecked with numerous fragments of pigment, the usual legacy of an attack of iritis.

L. eye:—Fringe of iris extending from 8 to 1.30 o'clock. A slight central anterior capsular opacity present. Fundus and Tension—normal.

IV 59-75. All normal eyes.

V1 and V2. Normal eyes.

V3 D. W., aged 2 years.

R. eye:—complete aniridia. Lens is completely clear: equator of lens can be seen all round. Fundus appears normal, but disc and macular area difficult to see on account of child's restlessness.

L. eye:—As on R. side. A slight central opacity is present on anterior capsule of lens.

V4-7. Normal eyes.

V8. M. S., aged 4 years. This child is troubled by photophobia in bright light. She has a R. convergent squint, which has been present for two years.

R. eye:—Iris present, though narrower than normal iris and with a crenated pupillary margin, between 7.30 p.m. and 5 o'clock. Lens is not dislocated, but lower edge can be seen below. Fundus—normal. Vessels tortuous.

L. eye:—Large coloboma iris between 5.30 and 7 p.m. Lens in normal position and lower edge visible.

Precise vision not determined: each eye has a refractive error of +3.50 D. sph. and +1.00 D. cyl. at 90°.

V9-V20. Normal eyes.

I am very much indebted to Mr. Esmond Recordon for introducing me to this family and for his generous encouragement and advice. Dr. R. R. Race very kindly did the blood examinations.

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INDUCTION OF AN EXPERIMENTAL TUMOUR OF THE LENS*

BY

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LONDON

It is a curious fact, hitherto ignored by pathologists, that neoplasms of the lens are unknown to ophthalmologists. It is often stated by students of cancer that any tissue or organ capable of cell division may be the site of a malignant neoplasm, yet the lens, as far as we know, in spite of the fact that mitoses occur in the subcapsular epithelium throughout life, never produces a cancer. This immunity may theoretically be due to various causes. It might be that the cells of the lens vesicle are inherently from the beginning resistant to malignant change. This would be strange since, if it were true, the lens would stand practically in a class by itself. It might also be that the presence of the capsule inhibits new growths of the lens, either by the tension it exerts or by its properties of a semipermeable membrane, preventing access of the required stimulus to the lens cells. That this is not the case is obvious, since lenses with ruptured capsules never become malignant. A third possibility is that the lens owes its immunity

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to its lack of blood supply. This presupposes the possibility of a blood borne cause of cancer. It would therefore seem of interest both ophthalmologically and pathologically to discover whether the cells of the subcapsular epithelium (the only ones capable of dividing after birth) could be induced experimentally to undergo malignant change.

Pathological techniques for the induction of experimental cancer include the use of chemical carcinogenetic agents and the investigation of inbred strains of animals. By a combination of these methods applied to the lens it can be shown that, when treated with a chemical carcinogen *and* provided with a blood supply the subcapsular epithelium will produce an epithelioma of extreme malignancy. There is, therefore, no inherent intracellular immunity of the lens to cancer and it is likely that its escape is due to its avascularity. The chemical agent chosen for the experiments was methylcholanthrene. This is known to induce tumours in many species of mammals (including rabbits and mice) when a minute quantity of it is placed in or in contact with the organ under investigation. Some strains, even of the same species of animal, are more resistant than others to this agent and in all species there is a latent interval of weeks, months or years (according to average length of life) before the tumours appear.

The first experiments were done on rabbits. A droplet of 0.4 per cent. solution of methylcholanthrene in liquid paraffin was injected with a fine needle into the lens cortex. The minute puncture of the capsule sealed completely and the lens remained clear, so that slit-lamp observation was possible. The oil could be seen lying in globules along the needle track. It showed a purple fluorescence in the slit-lamp beam, owing to the presence of the methylcholanthrene. The eyes were observed at weekly and then monthly intervals for 10 months, but no change in the lens occurred. Transparency was maintained and the droplets did not change their position in the lens, though a few minute crystals separated out within them. The eyes remained quiet throughout. It is possible that the animals have not yet been observed sufficiently long, but so far no effect has been produced by injecting the lens *in situ*.

It therefore seemed advisable to attempt to transfer the lens to a position where it could acquire a blood supply and there to treat it with the methylcholanthrene. For this purpose inbred strains of animals are necessary, and mice were chosen, both for their relative rapidity of reaction and because inbred strains showing a high natural cancer incidence were available.

Homozygous strains of mice are produced from a single pair by brother-sister matings through many (at least 30 and preferably

100) generations. When successful this inbreeding produces a pure strain in which all the mice resemble each other, all react in the same way to the same stimuli and all can be grafted with tissues (both normal and malignant) from each other without the production of immunological reactions leading to the death of the graft. Many of these strains exist and one of them, the C3H strain, was chosen as it was naturally prone to develop cancer and was known to react readily to methylcholanthrene.

The method was suggested by the work of Peyton Rous and Smith¹, who showed that when embryo tissue of pure line strains was grafted intramuscularly in adult related mice together with a trace of methylcholanthrene, malignant teratomas developed. These could be maintained indefinitely by transplantation from mouse to mouse of the same strain.

The lenses were dissected out from the eyes of young mice and implanted with the carcinogen under the skin of the flank of adult related mice. Three cancers of subcapsular epithelium were obtained.

Experiments and details of technique

Thirty-eight experiments were performed and 26 tumours resulted. Of these, three were carcinomas of the subcapsular epithelium and the remainder various types of sarcoma of the host mouse or epitheliomas of its skin. The three lens tumours occurred in the subcutaneous tissue of the flank of male mice, which rules out any possibility of derivation from mammary gland tissue of the host. The lenses were obtained from the eyes of mice of various ages (new born, two days, three days, four days, seven days and nine days old), but tumours were induced only with seven day and nine day old lenses. They were inserted under the skin of the flank of mice of the same inbred strain, together with methylcholanthrene. In some cases the hyaline capsules were ruptured and the lenses mixed with a few crystals of methylcholanthrene. In others the unruptured lenses coated with a solution of the carcinogen in oil were used. In others again teased lenses were mixed with methylcholanthrene in solution in equal parts of soft and liquid paraffin. The exact method employed did not appear to be important, two of the tumours being obtained with a solution of 1 mg. of methylcholanthrene per 0.5 c.c. of a mixture of soft and liquid paraffin, and the third tumour with crystals only. In all the successful experiments the lens capsules were ruptured. The tumours appeared between two and three months after implantation.

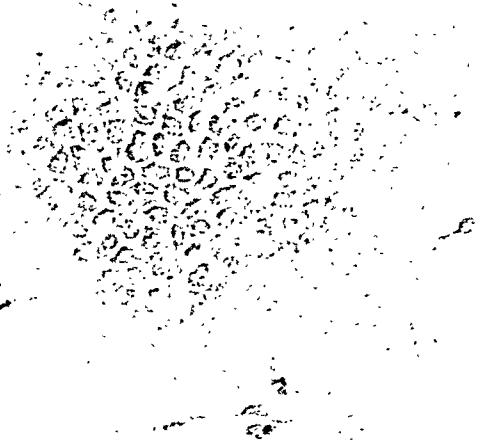


FIG. 1.

Tangential section through the subcapsular epithelium of the mouse lens at the anterior pole.

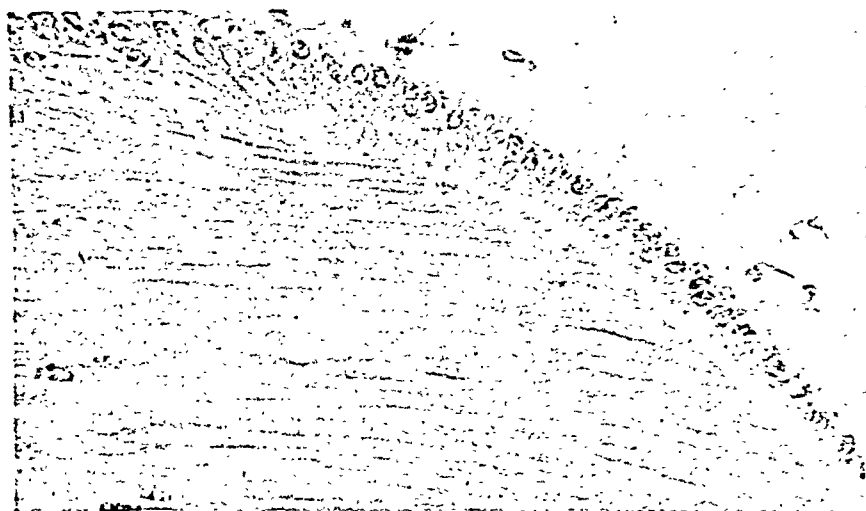


FIG. 2.

Subcapsular epithelium and equatorial region of lens of seven day old mouse.

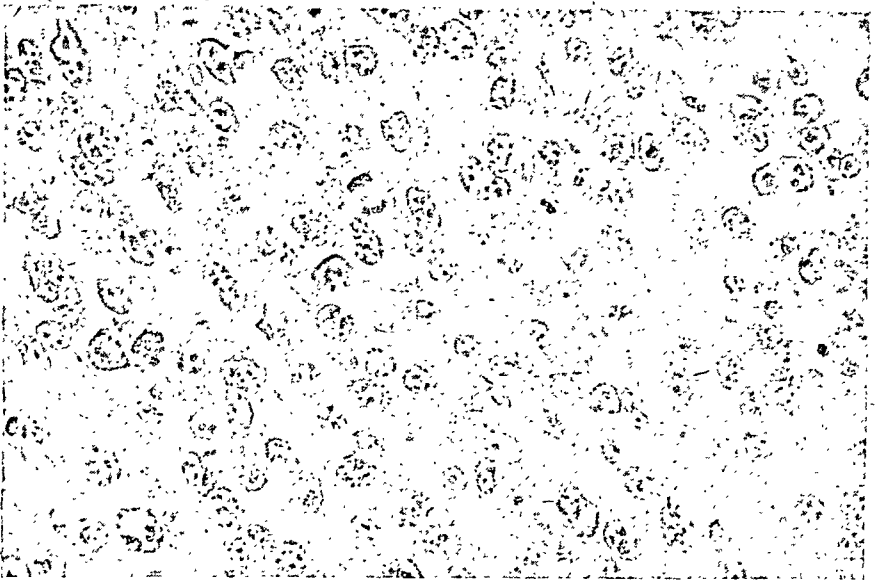


FIG. 3.

Section through lens tumour. Nuclei showing mitosis and typical arrangement of chromatin.

The tumours obtained were anaplastic carcinomas possessing certain characteristics of structure suggestive of their origin from the subcapsular epithelium (Figs. 1 and 3). They were investigated by stained sections, by smear preparations, by passaging them through other mice of the same strain and by observing their behaviour in tissue culture.

The first tumour obtained (known as M8.134) can be taken as the type and described in full.

Protocol of experiment M8.134

The lens of a seven day old C3H mouse was mixed with a 0.2 per cent. solution of methylcholanthrene in equal parts of soft and liquid paraffin and inoculated into the subcutaneous tissues of the right flank of a male C3H mouse one month old. The small lump resulting from the inoculation remained for three—four weeks and finally disappeared. A tumour appeared at the site fairly suddenly about two and a half months after inoculation and grew rapidly, being ready for transplantation a fortnight later. The mouse was killed and the tumour examined and transplanted into six C3H mice. It has retained its characteristics through eight

passages so far and continues to grow rapidly, no failure being noted in any of the transplants.

Characteristics of lens tumour M8.134.—The tumour tissue is greyish and translucent. The grafts grow rapidly and form large tumours filled with necrotic tissue and blood. Sections of the periphery of the tumour show large cells with voluminous or oval

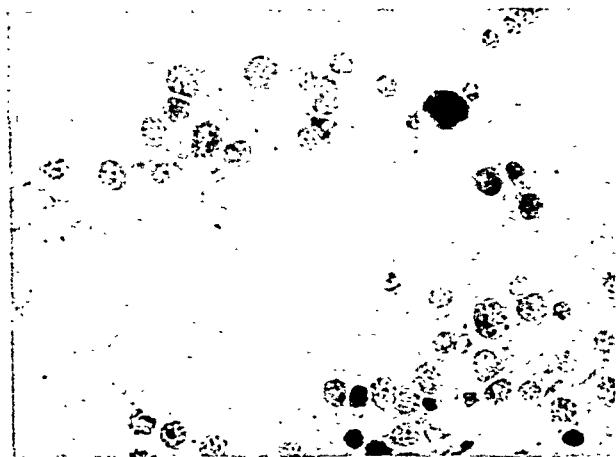


FIG. 4.

Smear preparation of lens tumour. The typical chromatin arrangement and the displacement of some of the nuclei to one side can be seen.

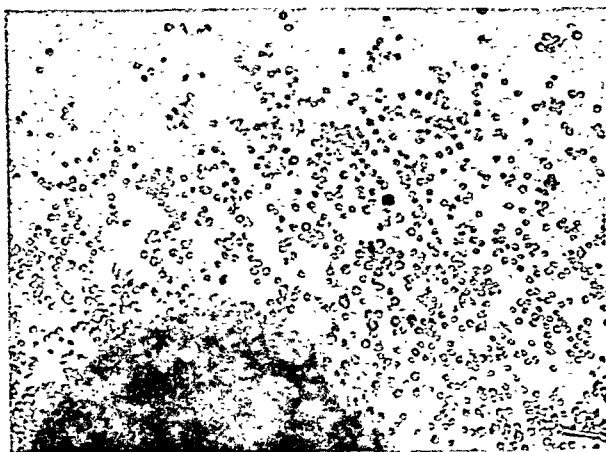


FIG. 5.

Tissue culture of lens tumour at 48 hours. Living preparation.

nuclei, prominent nucleoli and a reticular or punctate arrangement of chromatin. They resemble the normal cells seen in Fig. 1. Mitoses are frequent. In the young cells the nucleus is central, in the older cells it moves to the edge of the cell just before becoming pyknotic, as does the nucleus of the normal subcapsular epithelial cell before it differentiates into a lens fibre (Fig. 2). Large areas of necrosis and liquefaction occur in the centre of the tumour. Metastases occurring in the inguinal lymph nodes show the same

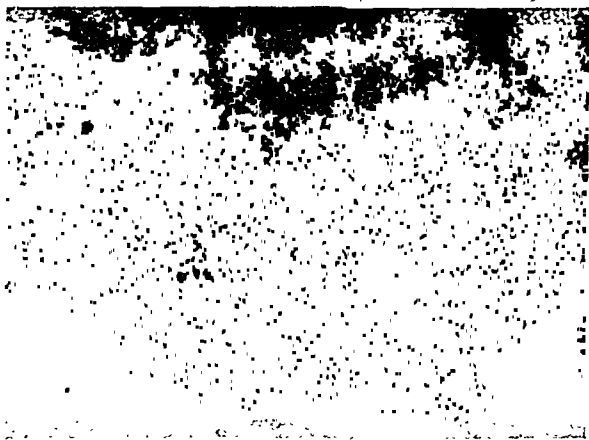


FIG. 6.

Extending flat sheet of cells at edge of tissue culture of living lens tumour at four days.

structure, but with less necrosis. The tumour is soft and the cells can be well seen in smear preparations (Fig. 4).

The tumour was grown in tissue culture* and Figs. 5, 6 and 7 show some results. Fig. 5 shows the explant after 48 hours. Tumour cells and a few fibroblasts are beginning to wander out. By four days (Fig. 6) the tumour cells have arranged themselves in the flat sheet typical of tissue cultures of carcinomas, as distinct from sarcomas, which show a scattered arrangement. If the culture is continued the sheet of cells tends to break away from the tumour explant and large and small islands of cells float free in the medium. Fig. 7 shows this and also demonstrates clearly the epithelial character of the cells. A high power photomicrograph of a portion of the sheet in Fig. 7 is seen in Fig. 8. The preparation is fixed and stained. Mitotic figures and completed mitoses are

* The best medium was found to be one part of rat serum, two parts tyrode solution and one part mouse embryo extract. Sometimes a few drops of distilled water were added.

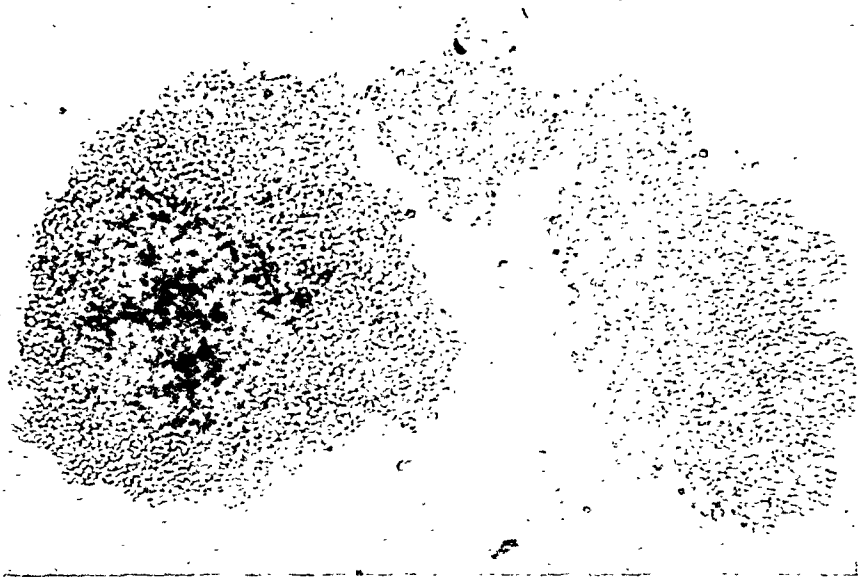


FIG. 7.

Five day tissue culture of lens tumour M8. 134. The explant is on the left, the new sheets of cells are breaking away on the right. Living preparation.

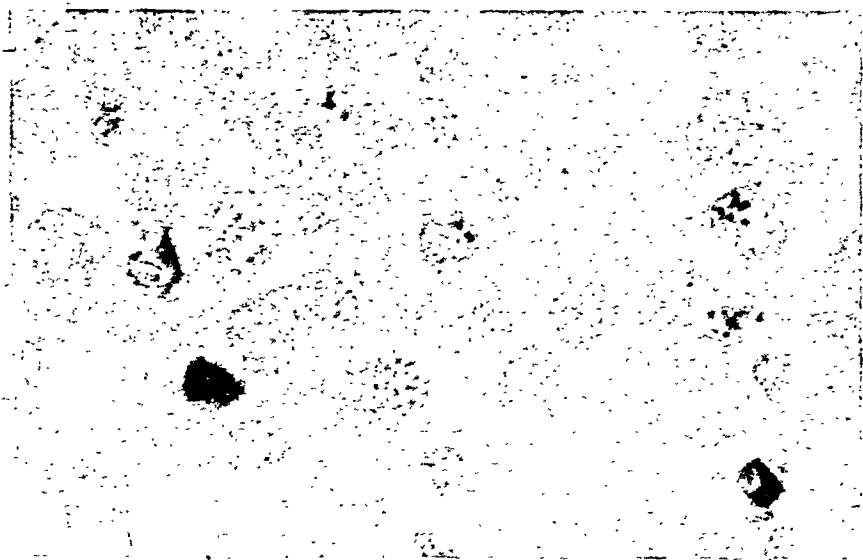


FIG. 8.

Fixed and stained preparation of five day tissue culture of tumour M8. 134. Note characteristic nuclei and presence of mitotic figures.

seen. The similarity of the cells to those of the tumour seen in Figs. 3 and 4 is apparent. In some of the tissue cultures of the tumour the extending edge showed fibrillar outgrowths somewhat similar to those described by Kirby² in tissue cultures of chick lenses. These are seen in Fig. 9. This fixed preparation of a five day tissue culture of the same tumour also demonstrates the attempted differentiation by movement of the nucleus to the side of the cell before pyknosis sets in.

The other two tumours resembled M8.134 in their structure and behaviour. They were slightly slower in growth and one of them, although showing the same characteristic epithelial cells, also contained sarcomatous areas, probably derived from the host.

The main points which show these tumours to be derived from the implanted lens epithelium are their carcinomatous character, their growth in tissue culture in friable sheets and the displacement of the nuclei and their degeneration in the course of attempted differentiation.

Since these tumours are derived from the lens epithelium which is the anlage of the lens fibres, one might expect them to resemble these somewhat in chemical composition and therefore to contain



FIG. 9.

Growing edge of five day tissue culture of lens tumour M8. 134 showing fibrillar processes from the cells and the attempt at differentiation shown by the movement of the nucleus to the side of the cell. Some of these displaced nuclei are pyknotic.

the organ specific protein α crystallin. Although this is by no means the only protein in the adult lens, its universal presence has led to the idea that the lens, unlike all other organs, has very little or no species specificity. If this were true (which is doubtful) these lens tumours might be expected to grow when grafted into other strains of mice, or even into other species of animals. Experiments to decide whether this was the case were therefore undertaken. The tumours, being known to grow well when grafted into mice of the same homozygous strain, were grafted into mice of other inbred strains, some showing a high and some a low incidence of spontaneous cancer. The high cancer lines, Strong 3 and R3 and the low, C57 black and the S strain, were used. The tumour failed to grow in all of these. It is not, therefore, even chemically common to mice as a whole.

The work of Greene³ on the growth of heterologous tumours in the anterior chamber of the rabbit's eye suggested that the mouse lens tumour might be tried in this way. Two of the three lens tumours were planted out on to the iris of rabbits, and the course of events carefully watched with a slit-lamp. A slight inflammatory reaction occurred around the tumour, shown by a gelatinous exudate and a few haemorrhages. The graft then shrank, became buried by the iris stroma and disappeared. In 10 days its site was only apparent as a small puckered scar-like area on an otherwise normal iris. Lens tumours do not, therefore, exhibit organ specificity.

In conclusion I wish to thank the Council of the Imperial Cancer Research Fund for their assistance in this research.

Summary

The statement has been made by general pathologists that any tissue or organ capable of cell division may be the site of a malignant neoplasm. Ophthalmologists, however, agree that carcinoma of the lens is unknown, although mitoses occur in the lens epithelium throughout life. The possible reasons for this are discussed. Experiments showing the induction of cancer of the lens in mice are described, using pure line strains and chemical carcinogens. These demonstrate the ability of the lens epithelium to become malignant under certain conditions, and throw some light on the immunity of the organ in the body.

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XANTHELASMATA AND AUTONOMIC NERVOUS SYSTEM. A NEW SYNDROME*

BY

A. BAKKER

GRONINGEN

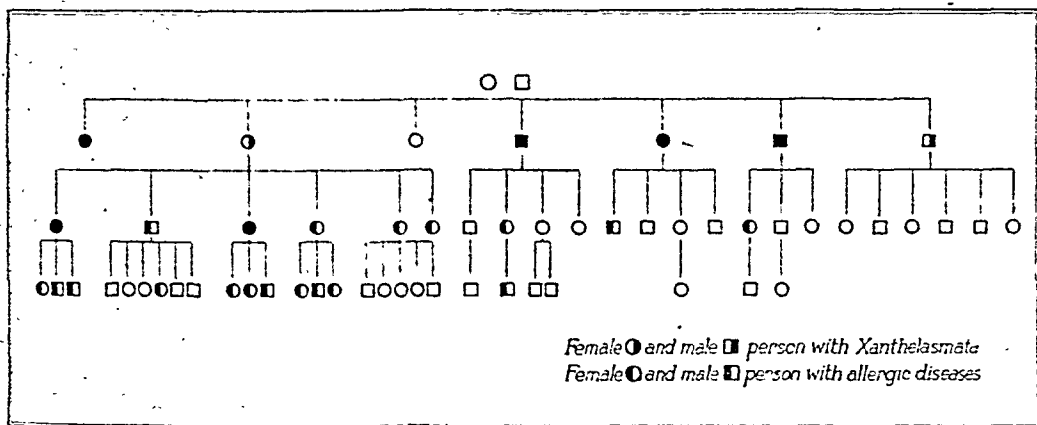
THE common opinion on xanthelasmata is that these small tumours, which are hardly elevated above the level of the skin, are produced by local degenerative processes.¹ Predilection place is the skin of the eye-lids. They are quite harmless; the only trouble they cause is a slight disfigurement of the face, which, however, evidently does not disturb everyone. At least, I know ladies who were not aware of the fact that they were bearers of these tumours. Generally these new growths are only mentioned casually and it does not often occur that patients call on an oculist expressly for the purpose of getting rid of them.

When one's attention is fixed on the existence of these tumours, one gets the impression that they occur more frequently than one originally expected. The reason that I am giving special attention to the xanthelasmata is the accidental circumstance that I had to treat several members of one family who were not only bearers of these tumours, but were also suffering from various kinds of allergic diseases, such as asthma, hay fever, urticaria, eczema and migraine (see pedigree).

When patients with xanthelasmata are carefully interrogated, they, themselves, show almost without any exception one or more types of allergy and, if they do not, other members of the family do. However, it is necessary to interrogate all these members personally, as it often occurs that for instance one sister does not know that her other sister has xanthelasmata or allergic symptoms. The following case gives a good picture of the intimate relation which exists between the apparently different kinds of these diseases in one family. A lady with xanthelasmata gets urticaria after eating strawberries or after taking aspirin. In two of her sisters urticaria breaks out after contact with a primula obconica. Another sister has hay fever. A son of her mother's brother is suffering from allergic asthma.

I have not sufficient statistic material at my disposal, thus I am not quite certain, but I got the impression that also an arcus senilis often occurs simultaneously with xanthelasmata at a relatively early age.

* Received for publication, March 25, 1947.



We may say that the frequency of the above mentioned allergic diseases is rather great, but doubtlessly not so great that it can give an explanation for the simultaneousness of xanthelasmata and allergic disorders.

From the pedigree it is visible that the inheritance of the allergic symptoms is of a dominant character. Of the great-grandmother and the great-grandfather no peculiarities were available. Four of the seven children of these ancestors show the syndrome (the third child died at a relatively early age). The remaining six children, all still living, have xanthelasmata. Unfortunately I had not the occasion to examine nor to interrogate the offspring of the seventh child and thus I am not certain that these individuals have really no symptoms at all. The same can be said of the five children in the youngest generation, whose parents wrote me that they are unaffected. Among the members of the third generation we only find two persons with xanthelasmata. This is, however, not astonishing, because most of them have not yet reached the age at which these tumours generally appear.

It was striking to observe that many of the patients with xanthelasmata had a labile autonomic nervous system and this may perhaps supply the key to the obscure secret which envelops the relation between the xanthelasmata and the allergic disorders. It is a well known fact that also allergic patients often show various kinds of dysfunction of the vegetative nervous system. One of the male representatives (aged 45 years) in my pedigree possessed a whole series of these symptoms, such as hyperhidrosis, an infantile respiratory arrhythmic pulse, cold feet, blushing and other vasomotor disorders and spastic constipation.

If we now ask the question whether there is anything known of the influence of the sympathetic or para-sympathetic nerves on the

developing of xanthelasmata we must answer that little or nothing is known of it. These tumours are built up by large, epithelium-like cells with spongy protoplasm. These cells contain large quantities of neutral fat and cholesterol. Corresponding to the above described close relation which exists between these tumours and more generally determined somatic disorders, it seems quite improbable that the aetiology of xanthelasmata must be looked for exclusively in local degenerative processes. Also the results of several investigators who noticed a hypercholesterolemia in these patients is not in accordance with the latter opinion. Certainly, not every bearer of xanthelasmata shows an increased concentration of cholesterol in the blood, but this does not prove that in some period of his life he has not had it. Furthermore we must pay attention to the ratio of free cholesterol and its esters with unsaturated fatty acids. This ratio seems to be of especially great importance and a disturbance of this normal ratio we could call dyscholesterolemia.

Our knowledge in questions of fat and lipid metabolism is rather defective. Cholesterol in every respect is a curious substance. We may say that generally the animal organism is not able to synthesize cyclic products (acyclopoiesis), but the cholesterol-synthesis is an undeniable fact. Nevertheless, a large fraction of the required cholesterol is taken in with our food. Before the resorption in the intestine can take place both fats and lipoids have to undergo phosphorylation. The investigations of Verzár² have shown that corticosterone katalyzes the processes of phosphorylation and here, perhaps, we have the connecting link between the autonomic nervous system and the cholesterol metabolism. Experiments of de Langen³ probably point to the same direction. Hyperlipaemia in rabbits was caused by artificially provoked anaemia. No hyperlipaemia developed when the spinal cord between the third and fourth thoracic vertebrae was cut.

It would be of great interest to know more about the fluctuations in the cholesterol concentration in the blood of patients with xanthelasmata and this especially during long periods of their life. Likewise it would be useful to have more information on the lipid metabolism in patients with allergic diseases. But as far as I know there are no investigations dealing with this subject. Theoretically we must finally take into account the possibility that an increased cholesterol content of the blood has influence on the tonus of the sympathetic and para-sympathetic nerves and the result of this could be a hypersensitivity for substances such as albumen, pollen, strawberries and so on.

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Summary

Almost without exception patients with xanthelasmata suffer from various kinds of allergic diseases, and if they do not, at any rate we find allergic disorders in their relations. As these benign tumours are probably closely related to the cholesterol metabolism and the latter stands under the regulating influence of the autonomic nervous system and as finally allergic disorders have much to do with the sympathetic and the para-sympathetic nerves, an attempt is made to consider all these apparently heterogeneous diseases from one viewpoint, *i.e.*, a dysfunction of the vegetative nervous system.

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A CASE OF BILATERAL COLOBOMA OF THE OPTIC DISC*

BY

H. L. HUGHES

LONDON

Report of a Case

THE patient, Patrick N., aged 24 years, presented with the complaint that his right eye had turned outwards since an injury when he was only a few months old. It had not troubled him until he contemplated marriage and he came to ask for a cosmetic operation. (The patient was demonstrated at the May, 1947, meeting of the Section of Ophthalmology, Royal Society of Medicine).

Family history and past medical history: essentially irrelevant.

Examination. Vision: Right eye P.L. only. Left eye 6/6.

Under H. and C.

$$\begin{array}{r} -5.0 \\ | \\ \hline | \\ -4.5 \\ | \end{array}$$

Not improved \bar{c} lenses.

Not improved \bar{c} pinhole.

$$\begin{array}{r} +1.25 \\ | \\ \hline | \\ +1.25 \\ | \\ +0.5 \\ \hline +0.25 \text{ at } 90^\circ = 6/5 \end{array}$$

* Received for publication, July 2, 1947.

The right eye shows a divergent strabismus with an angle of 40° .

The eye movements are full, but the patient cannot fix with the right eye. There is no nystagmus. The eyes are of normal size. The pupils are round, regular, equal and react to light and accommodation. The right pupil reacts more sluggishly than the left.

The right cornea shows evidence of past inflammation in the form of both superficial and deep vessels. The left cornea is clear.

Ophthalmoscopic Examination, Figs. 1 and 2.

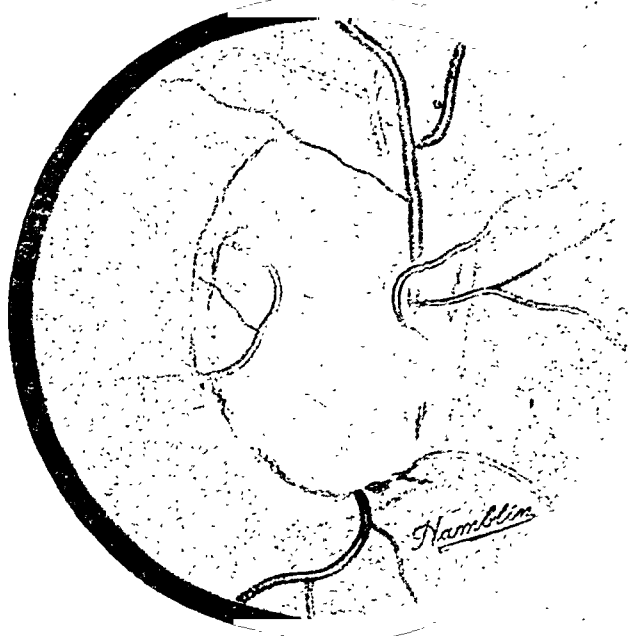


FIG. 1.

Right eye. The most striking part of the fundus picture is the pseudo-optic disc which appears to be two and a half times the normal size. It is deeply cupped and of a dead white pallor.

The upper half is rounded and well outlined and contains a rim of pink nerve tissue. The lower half is deeply cupped. The retinal level at the lower border of the disc is seen with a -1.0 D. whilst the floor of the cup may be defined with -18.0 D. The entire pseudo-disc is bordered by scattered pigment and on the nasal side is a white scleral ring.

The vessels in the upper half of the disc emerge from the pink nerve tissue within the disc surface, whilst below they dip over the steep edge of the cup and are lost to view.

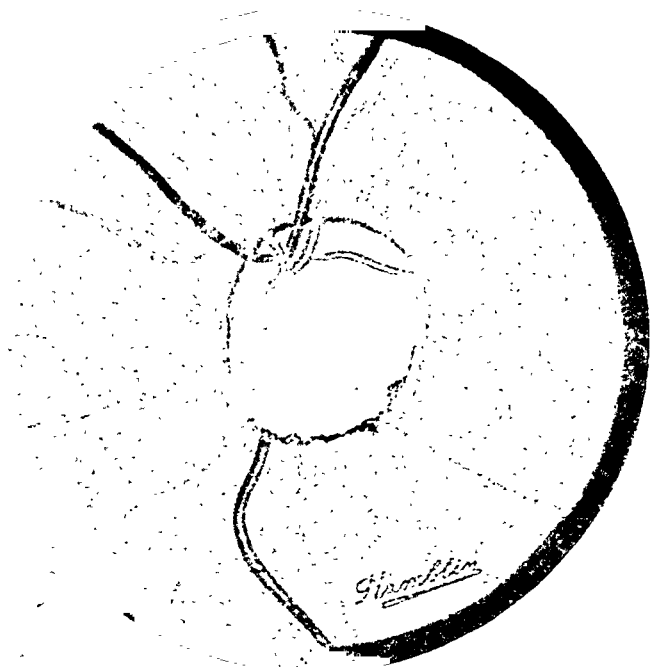


FIG. 2.

Left eye. The condition is similar but not as marked. The retinal level at the lower border of the disc is seen best with $+0.5$ D. and the floor of the cup with -6.0 D. The vessels behave like those in the other eye. The intra-ocular tension is:—

Right 20 mms.	} Schiötz
Left 22 mms.	

There is a suggestion of a crater-like hole at 2 o'clock.

General examination revealed no other developmental abnormalities.

Discussion

The case is presented as an addition to the 50 odd cases recorded and recently reviewed by Steinberg of San Francisco.

The condition is usually unilateral when vision may be normal but is more often seriously defective. Bilateral cases have been recorded by Adler, Calhoun, Johns, and more recently by Magnus. In such cases one eye is usually blind whilst the vision in the other is nearly normal.

The most generally accepted theory of pathogenesis is that of von Ammon—"defective closure of the optic cup in its extreme posterior position. Associated is an abnormal development of the

surrounding mesoderm (precursor of choroid and sclera) with a later extension of the resulting scar tissue."

Johns offered as explanation a failure in development of the papillo-macular bundle the fibres of which would normally occupy the area of the cup. Such an explanation is inconsistent with vision such as that of the present patient, *i.e.* 6/5 (left).

The classification of such cases usually follows that given by Caspar who described three groups.

(1) Cases in which all the vessels are coming from the lower portion of the pseudo-disc, even those which later turn upwards.

(2) Cases in which vessels emerge at or a little above the centre—their arrangement being almost normal.

(3) Cases in which the vessels appear at the circumference of the disc and appear to bend sharply around its edges.

The present case falls into the third group. The diagnostic features are: (1) Enormous size of disc, or what corresponds to disc. (2) Deep cup. (3) Dead white pallor.

The differential diagnosis must include for completeness: (1) Glaucomatous cupping. (2) Cavernous optic atrophy.

The first is excluded by a study of tension and fields. The latter occurs at a much later period.

I am indebted to Prof. Arnold Sorsby for the diagnosis of this case.

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A CASE OF PSEUDO-GLAUCOMA

BY

J. A. MAGNUS

YORK

CASES showing typical glaucomatous cupping, but without increased intra-ocular pressure, have always aroused general interest. The various authors give them different names: amaurosis with excavation (v. Graefe), glaucoma without hypertension, primary cavernous optic atrophy (Schnabel), but I think Duke-Elder is right when he suggests that these terms should not be used. He calls these cases pseudo-glaucoma.

The following case is of interest because calcification of the internal carotid artery could be shown radiologically.

Robert Henry B., aged 74 years, was first seen on November 23, 1945. He had no complaints of anything in particular, he just wanted new reading glasses.

On examination the vision of the right eye was:—

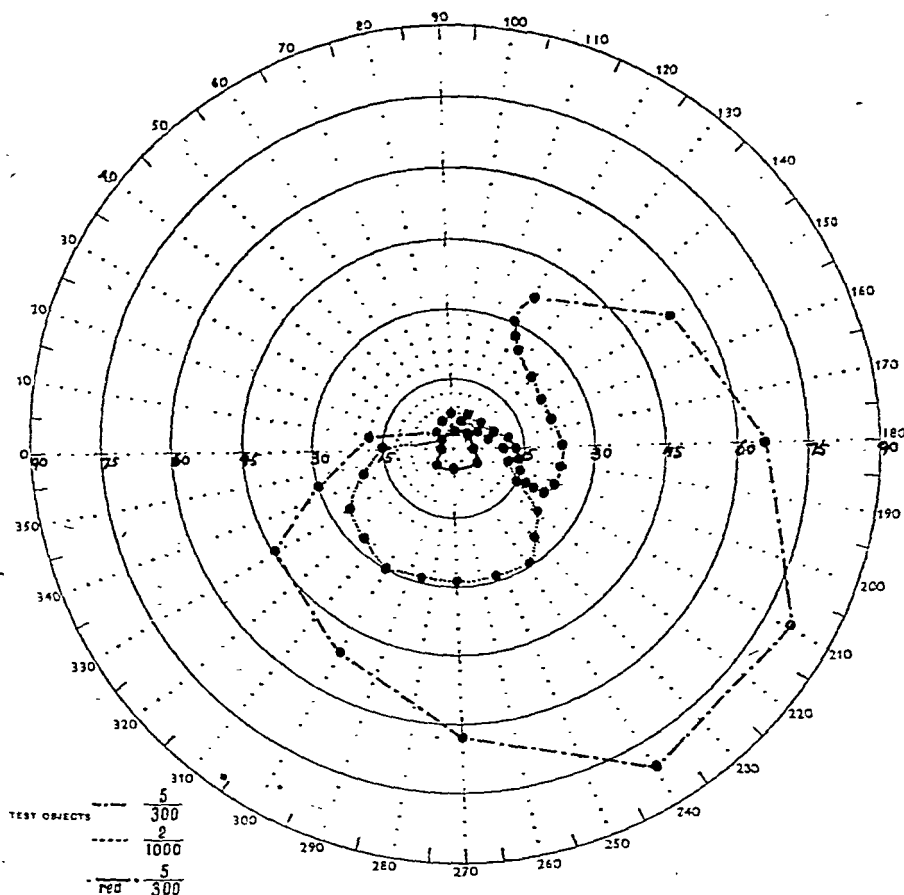
With $\frac{+0.5}{-3.5 \downarrow 90^\circ} = 6/9$ to $6/6$ (2 letters)

that of the left eye:—

With $\frac{-1.75}{-2.75 \downarrow 90^\circ} = 6/9$ (3 letters)

With the addition of $+2.50$ reading small print (J.2).

RIGHT EYE



LEFT EYE

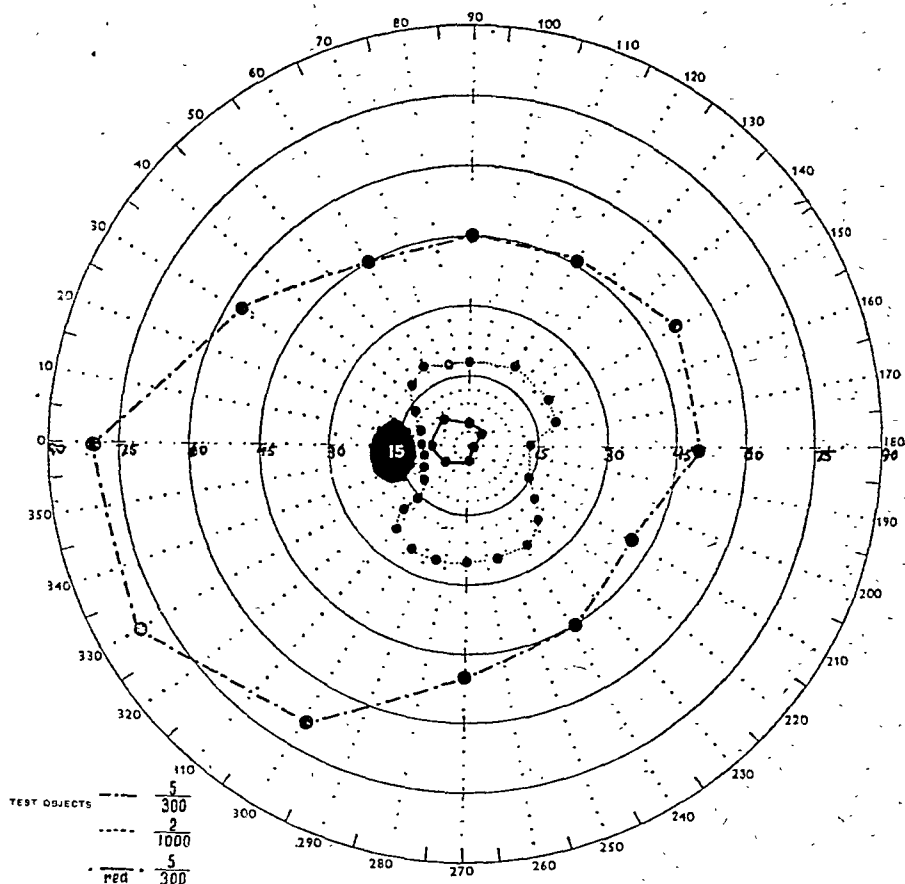


FIG. 1.

Both fundi showed deeply cupped discs, which were slightly pale.

The blood pressure was 160/80, and the intra-ocular pressure $\frac{5.5}{6} = 19$ mm. Hg in both eyes. This pressure did not rise after one hour's stay in the dark-room, nor after repeated instillation of homatropine for one hour.

The right field showed marked nasal step, reaching the fixation point, and a typical arcuate nerve fibre bundle scotoma; the left one general peripheral depression. The field for red was, in both eyes, disproportionately smaller than the field for white. It is of the atrophic type (see Fig. 1).

FIG. 2.



See Schema on p. 695, Fig. 2.

The X-ray of the sella shows calcification of the internal carotid artery (see Fig. 2).

The question arises—is this case a glaucoma? The difficulty of the diagnosis lies in the fact that the fundi and the field defects appear to suggest this disease. The radiological finding is, therefore, of great importance, because it furnishes us with a reasonable explanation for the field defect, because the intra-ocular pressure was always found quite normal, even after provocative tests. The calcified internal carotid pressing on each temporal side of the chiasma has caused the nasal contraction, the nerve fibre bundle

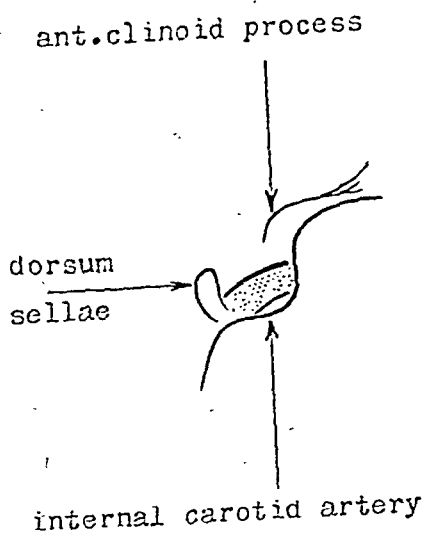


FIG. 2.

scotoma being most likely due to atrophic changes in the optic nerve itself.

Pickard pointed out that in glaucoma the fields for white and colours are similar, since all conduction is equally destroyed by pressure, but that in cases of glaucoma without hypertension, due to cavernous optic atrophy, the field for red shows a general contraction not in proportion to the contraction for white objects. The above case shows this disproportion of the fields.

It is difficult to find a reasonable explanation for the cupping of the discs, unless one assumes either a direct damage of the nerve fibres through pressure of calcified blood vessels, or areas of degeneration and softening within the optic nerve itself, through end-arteritis or spastic contraction of the nourishing blood vessels. If these areas of degenerated nerve fibre tissue are situated just behind the lamina cribrosa the latter will recede, and produce a glaucomatous cupping.

Redslob published in 1941 a number of cases of glaucoma without increased intra-ocular pressure, where the histological examination revealed typical cavernous atrophy of the optic nerve. These cases showed extensive vascular changes, due to venous stasis and Redslob thinks that the vascular upheaval is the responsible cause for the formation of the cavernous atrophy and the glaucomatous cupping.

Weekers, on the other hand, speaks of incomplete glaucoma. He says:—Glaucoma originates chiefly from a change in the intra-ocular blood vessel system. In the case of chronic glaucoma, *i.e.*, idiopathic glaucoma, the vascular alteration affects the uvea, the retina and the optic nerve, causing the three principal symptoms of glaucoma:—increased intra-ocular pressure, narrowing of the field of vision, and glaucomatous cupping of the disc. The three symptoms are usually, but not always, found simultaneously. There is an incomplete glaucoma: in certain cases the increased intra-ocular pressure may be absent. The incomplete glaucoma is observed in grown-up and aged patients, whose vascular system is more or less defective and the affection progresses very slowly. The cupping of the disc may remain isolated for a long time, but may, in course of time, be complicated by field defects. Occasionally hypertension may appear. These accompanying symptoms show that the lesion of the optic nerve must be attributed to glaucoma. There are a series of intermediate stages between monosymptomatic cupping without hypertension and complete glaucoma with ocular hypertension.

Taking all the various points into consideration I feel strongly that the above case does not fall into the category of monosymptomatic glaucoma, but that the calcified internal carotid artery proves a vascular cause. In favour of this are the atrophic type of the field changes, and the perfectly normal intra-ocular pressure, in spite of provocative tests. The field and fundus changes have remained stationary for over one year, a fact stressed by Thiel, who gives these cases a good prognosis.

Summary

A case of pseudo-glaucoma is presented, which shows field changes, deep cupping of the disc, normal intra-ocular pressure, and calcification of the internal carotid artery.

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A CASE OF AN ADENOMA ARISING IN A SWEAT GLAND OF THE UPPER EYELID*

BY

F. CLIFTON and W. H. GORDON

LONDON

REPORTS of adenomata of the sweat glands are perhaps sufficiently rare to justify the description of a case where features are thought to indicate some points in the clinical diagnosis of these tumours.

Mrs. A.B., aged 70 years, seen recently as an Out-Patient, presented for examination a tumour of the left upper lid which had been increasing in size slowly over a period of five years. Clinical details of the early appearance of the lesion are not available, but it is known that the provisional diagnosis of ? sebaceous adenoma ? rodent ulcer, was made some time ago. The original lesion appears to have presented as a small crust on the skin of the lid near the inner canthus and adjacent to the margin.

On examination, there was present a rather flattened oval tumour with the long axis vertical, the lower pole of which overhung the lid margin, shielding also part of the caruncle from view. In size 1 cm. long and $\frac{3}{4}$ cm. broad, the smooth growth was covered by somewhat stretched though otherwise normal skin, movable over the tumour as a whole, but attached around the slightly raised edges of a small para-central crusted area. On palpation, the growth felt firm, yet not as hard as would have been expected in the case of an epithelioma. There was no regional glandular involvement and the tumour transilluminated freely.

Operation was performed by Mr. Eugene Wolff. A skin incision was made across the growth, which encapsulated in a thin fibrous sheath, was dissected out *en masse*, without difficulty. Small strips of redundant skin were removed from the margins of the incision which was closed by one silk suture. Healing was uneventful and rapid. The cosmetic result was very good, the lid appearing normal, save for the presence of a minute horizontal scar detectable on close examination.

Reference to the literature indicates that, in the past, extensive resections of the lid have been performed, with the necessity for subsequent plastic repair.

Pathology.—The skin is stretched over a tumour resembling in structure a tubulo-racemose gland. From the fibrous capsule, which is quite definite, except where the over-lying skin is

* Received for publication, June 4, 1947.

ulcerated, septa carrying well-formed vessels pass inwards to divide the tumour into lobules, and in turn give rise to finer strands supporting the branching tubules. In many places the fibres of this stroma are curiously swollen and ill-defined, occasionally so greatly that no structure can be made out, and the walls of many



FIG. 1.

Low power view showing the fibrous capsule and the numerous cysts.

of its blood vessels are similarly affected. The stroma is also infiltrated by lymphocytes which, here and there, form follicles.

Whilst in general the structure is tubular, in places there is proliferation of the parenchymal cells as solid masses, between some of whose cells cell-bridges can be made out. In these cell-masses are many degenerative cysts in all stages of development, from minute to quite large ones.



FIG. 2.
High power view of a cyst of type (1).

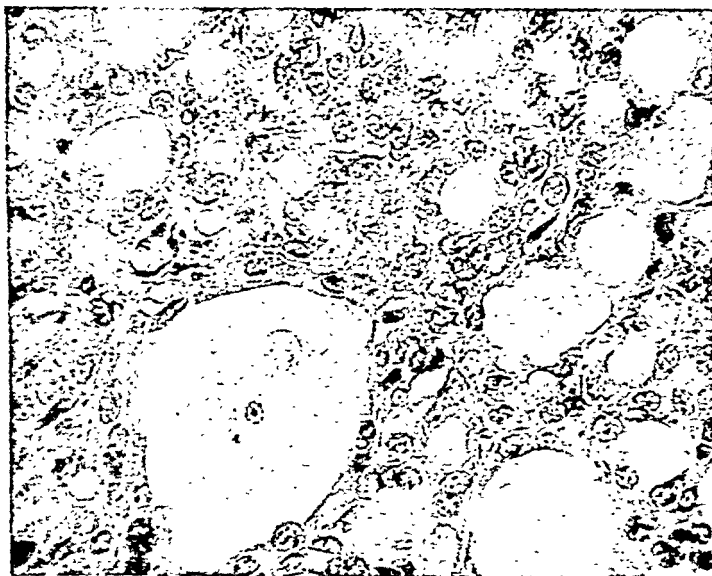


FIG. 3
High power view of cysts (pseudocysts) of type (3). Degenerated tumour cells are seen within these.

A section of the tumour shows spaces or "cysts" of the following three types :—

(1) Lumina of tubules lined by columnar epithelium similar to that of the normal sweat gland, and having sometimes also an outer layer of cubical cells with oval nuclei which probably represent the myoepithelial cells of the normal gland.

(2) Lumina lined by smaller altered gland cells having denser nuclei.

(3) Cystic spaces due to breakdown of tissue cells, lined by partly degenerated polyhedral cells or in places merely walled by the stroma, and containing an albuminous coagulum and occasionally disintegrating parenchymal cells.

Summary

A case of adenoma of a sweat gland has been described and it is suggested that if consideration be given to the long history, the presence of the crust, the mobility of the skin over the edges of the tumour, the translucency, and the firm consistency, perhaps best tested with the blunt end of a probe for cystic spaces, a clinical diagnosis of this condition should be possible.

AN UNUSUAL CASE OF SYMMETRICAL, BILATERAL, NON-TRAUMATIC IRIS PROLAPSE.

BY

M. L. NAIRAC

MAURITIUS

A NEGRO woman aged 22 years, was transferred from the island of Rodriguez to the Civil Hospital, Port Louis, Mauritius, on March 29, 1947.

I here quote the report of the Government Medical Officer who first saw the case.

"Two weeks before she was seen, on March 15, she felt a strong, burning pain in the right eye followed by a discharge of 'pus.' The following day the left eye became similarly affected. The pain was such that the patient could not open her eyes and in her own words was blind for a few days."

"When first seen the patient was suffering from intense photophobia. Both eyes were found to have a staphyloma in the upper quadrant, the right side greater than the left. The pupils were irregular and drawn up. The walls of the herniation were so thin

* Received for publication, May 26, 1947.

that they threatened to rupture. They were transparent, however, and showed no signs of injury or inflammation."

"She was married 5 years ago and has three children; no miscarriages."

"She was transferred to Mauritius on March 27, 1947."

I saw the patient on April 1, 1947, and found the following condition:—

Both eyes had an iris prolapse between 11 o'clock and 1 o'clock, through gaps which measured 6 mm. by 4 mm. in the right eye and 6 mm. by 3 mm. in the left eye. The upper parts of the irides were well prolapsed through the openings and formed bulges: in

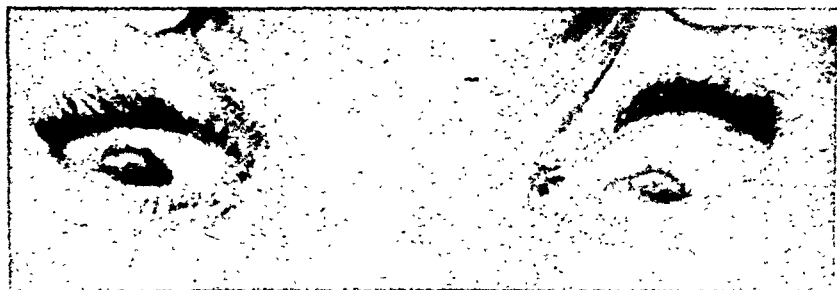


FIG. 1.

the right eye 6mm. \times 4mm. \times 3mm. and in the left eye 6mm. \times 3mm. \times 3mm. The pupils were drawn up towards the perforation more so in the right eye than in the left, but the edge of either pupil was not involved in the prolapse. The hole in each cornea was clear-cut, oval, and placed just central to the corneo-scleral junction. There were no signs of inflammation not even some ciliary injection near the prolapsed part of the iris. The rest of the cornea was bright and clear. The iris was normal in other respects; there were no posterior synechiae and its colour was normal. The anterior chamber below the prolapse was well-formed and the aqueous was clear. The fundi could be well-seen and were normal. There was no cupping of the discs.

Fig. 1 shows the condition, and although the photograph is not perfect it shows well the two bulges of the prolapsed irides, and the lack of inflammation. Kahn reaction was negative and there was no history of trauma. There was no evidence of avitaminosis. Atropine was instilled and the pupils dilated very well.

Treatment

It seemed obvious that it would be too great a gap to close with a conjunctival flap if the prolapsed part of the iris was abscised.

It was therefore resolved not to excise iris tissue but instead to cauterize it with the electric cautery along the whole of the prolapsed part and along the edge of the corneal hole, and then to apply a thick conjunctival flap which would thus adhere more readily.

On April 8, 1947, the left eye was done and the operation was carried out as planned.

On April 15, 1947, the flap having taken well, in the left eye, the other eye was similarly treated. But this time the iris was so thin that on the first impact of a not very hot cautery perforation occurred. We did not allow this to disturb the operation which was



FIG. 2.

carried out again as planned. The result was better than in the left eye.

Fig. 2 shows the condition after operation. This was taken on April 22, 1947.

On April 30, 1947, the patient was allowed out of hospital, and as far as one can tell has excellent sight and a strong conjunctival "patch" on either eye which should last her a long time.

The interesting problem here is one of aetiology. The probable explanation is of a bilateral acute infection, with the formation of corneal ulcers, which with the help of severe blepharospasm and probably a fair amount of rubbing perforated quickly and healed.

I do not think one can explain the condition in any other way. "Trophic" changes, avitaminosis, etc., would have shown other signs in the cornea and conjunctiva. These were lacking completely, nor was there any evidence of trauma. The curious thing is the symmetry of the lesions.

I would be glad to hear of any other similar case, or of any publication on the subject.

I am indebted to Dr. M. Shun-Shin, who first saw the case and brought it over Mauritius.

CONTRACTED SOCKET* †

The Splint Method of Post-operative Control

BY

F. MAXWELL LYONS

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THE traditional Wheeler operation, an admirable concept upon which most later procedures have been based, was primarily intended for the *obliterated* socket in which no useful conjunctiva remained and where complete relining of the reconstructed cavity with an epidermal graft was necessary. In the majority of distorted and contracted sockets resulting from civil and war injuries, however, a considerable amount of healthy conjunctiva persists. Whether this should be conserved or removed as a preliminary to grafting has long been a subject of controversy.

The widespread prejudice against mixing skin and mucous membrane on the grounds that it produces chronic irritation and discharge I have found to be largely unsubstantiated or, at least, over-stressed. On the other hand, the sacrifice of all existing conjunctiva has very definite disadvantages. Total relining, at its best, produces a capacious socket but one which lacks resilience and adaptability. The normal shrinkage of the graft adherent to the tarsal plates inevitably results in thickening of the lids and entropion which may be pronounced and, even in lesser degrees, detract from the final cosmetic effect.

In very few contracted sockets is there insufficient conjunctiva remaining to cover the tarsal plates, and for many years I have been convinced that this should be carefully preserved.

The greatest source of trouble in any form of socket reconstruction is post-operative shrinkage. As in all Thiersch grafting, about 30 per cent. shrinkage of the epidermal inlay must be accepted as "normal" and duly allowed for in planning the graft. Unless the bed is properly prepared and freed from scar tissue, this shrinkage will be much greater and little or no benefit may result from the operation.

In spite of all precautions and in the most favourable cases, post-operative contraction is seldom regular and, although the resultant socket may be adequate in size, its irregular shape will prevent the fitting of a standard artificial eye. The ordinary stent

* The acrylic moulds and the method of splint fixation described in this communication were demonstrated at the Meeting of the Egyptian Ophthalmological Society held in Cairo in March, 1946.

† Received for publication, June 3, 1947.

or acrylic mould with pressure bandage will not satisfactorily control this shrinkage and distortion. All of us must have witnessed at times the disheartening sight of rotation, tilting and even extrusion of the mould two or three weeks after an apparently successful operation.

The technique of grafting and post-operative splintage here described was designed to offer controlled resistance to the forces of contraction and to persuade the socket, from the start, to take

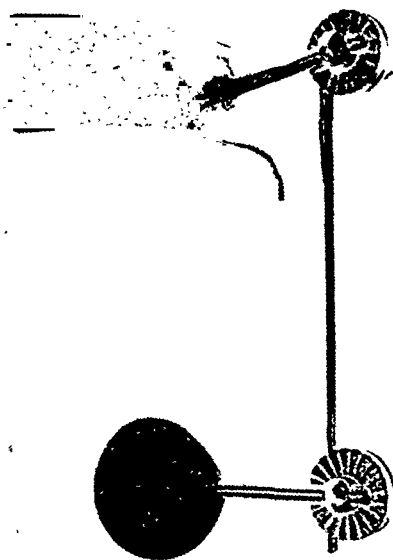


FIG. 1.

Showing arrangement of mould and the adjustable splint.

on and to keep the shape of a standardized prosthesis. It was arrived at by the gradual evolution of procedure and equipment in the course of a large series of cases undertaken during the war. In this I was greatly aided by the advice and practical help of colleagues—ophthalmologists, maxillo-facial surgeons, dental surgeons and mechanics.

Briefly, the epidermal graft is applied to the prepared socket in the usual way but on a special acrylic mould. At the first dressing (*i.e.*, on the 7th day) the mould is connected to a rigid but adjustable splint (Fig. 1).



FIG. 2.

Plaster-of-paris head-cap and splint in position.

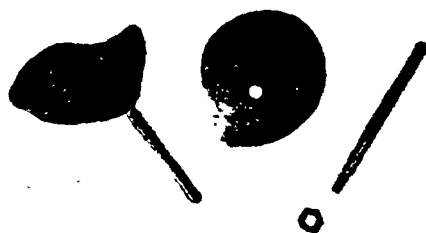


FIG. 3.

Showing details of mould and threaded rod. A nut similar to that shown in the photograph is embedded under the anterior surface of mould.

The fixed point is a frontal pin embedded in a plaster-of-paris head-cap and to this the mould is attached by stainless steel rods and universal clamps (Fig. 2).

The acrylic moulds are replicas of standard artificial eyes. Each mould has embedded below the anterior surface a small stainless steel nut (6 B.A.) into which a short length of rod, threaded at one end, can be screwed (Fig. 3). Latterly, Stallard's useful

suggestion—of drilling holes through the thickness of the mould to allow seepage of exudate and discharge from the back of the socket—has been adopted although it is not shown in the figures. A stock of 12 moulds, 6 right, 6 left, of sizes varying from medium to large covers almost all requirements.

Procedure

Anaesthesia.—General anaesthesia is preferable throughout the operation. Local anaesthesia is less satisfactory owing to the difficulty of blocking the nerves of the donor area of the upper arm. As a compromise, which has often proved useful, the dissection and preparation of the socket may be carried out under local anaesthesia and intra-venous pentothal given for the final stages of cutting and placing the graft.

Operation.—A wide canthotomy is first performed. The conjunctiva is then incised along a line corresponding with the lower border of the lower tarsal plate, extending from the outer canthus up to and behind the caruncle. Through this incision dissection is carried down to the infra-orbital margin, the periosteum of which is exposed throughout its length. This is an important step since the adhesions formed here help to anchor the lower fornix. Any redundant tissue is excised so that the tarsal plate covered with its conjunctiva may assume its normal vertical position. The caruncle should be preserved if possible.

The upper part of the conjunctiva is then freed from adhesions over the muscle stump and scleral remnants, if any. It is undermined and the upper fornix reformed, leaving the conjunctiva to cover the upper tarsus. Dissection here should be backwards rather than directly upwards and care must be taken to avoid injury to the levator muscle.

The conjunctiva should be delicately handled and any traction applied by holding-sutures rather than by forceps since pieces of graft may become adherent to the abraded surface and they are difficult to remove later. All fibrous bands must be carefully removed.

The largest mould over which the lids will close and which fits snugly into the lower fornix is selected by trial. It is removed from the socket, fitted with its short threaded steel rod and placed on one side ready to receive the graft.

Sutures for closing the canthotomy are inserted but left untied. All oozing is checked and the socket is lightly insufflated with penicillin and sulphathiazole powder. The area is covered while the graft is prepared.

A very thin epidermal graft, large enough to cover the mould and allow for overlapping, is cut. It is carefully eased off the knife into a bowl of warm saline so that it floats with its raw surface upwards. The mould, held by the attached rod, is insinuated underneath the graft and gently raised so as to lift the graft off the surface of the saline. With the aid of an iris repositor the graft can be evenly applied to the surface of the mould. If the graft is punctured over the posterior concavity of the mould it will readily subside into the slight hollow. Any redundant graft should be trimmed with scissors.

The socket is again examined to make sure that it is dry. Then, while an assistant retracts the lids, the graft and mould (still held by the rod) are lowered into the socket. The rod is unscrewed and removed, and the graft evenly spread over the anterior surface of the mould. The lids are closed and the canthotomy sutures are tied. It is not necessary to stitch the lids together although some prefer to do so. A tulle-gras and gamgee dressing is applied. Both eyes are covered with a firm crepe bandage.

The patient should be nursed flat or in a semi-recumbent position for the first two days; after that he may be allowed to sit up.

The dressings are left undisturbed until the 6th day. The bandages are then removed and the sound eye is uncovered. The dressing on the operated eye, if clean, is left *in situ* and fixed with adhesive strapping. A plaster-of-paris head-cap incorporating the metal frontal pin and plate is put on as shown in Fig. 2. First, a piece of stockinette is pulled over the head and over the ears. A single layer of crepe bandage is placed around the head and fixed with strapping; a similar piece of bandage is fixed across the top of the head in front of the ears. Next, a few layers of plaster-of-paris bandage are applied and reinforced over the frontal region. The pin and plate are now placed in position over the brow on the affected side and secured with several more layers of plaster bandage. For the sake of neatness, the stockinette is cut and folded back over the plaster and covered with a final layer or two of plaster bandage. It is left for 24 hours to harden. If the plaster is taken well down over the frontal region and under the occipital protuberance as shown in the figure, the head-cap will not lift off and will form a firm and steady support for the splint.

On the following day, *i.e.*, the 7th day after operation, the first dressing of the socket is done. The canthotomy sutures are removed. The lids are gently prised open and the rod is screwed into the hole in the mould. The mould is eased out of the socket. A pint of warm saline is poured over the socket and graft. Any pieces of redundant graft are removed. The excess saline is soaked up with a small piece of gauze. A few drops of penicillin (500

u/cc.) are instilled. The mould (with the rod attached) is dipped into sterile liquid paraffin and reinserted in the socket. It is then connected to the frontal pin by means of a suitable length of rod and two universal clamps. The position of the mould is carefully adjusted and the clamps are tightened with a spanner.

From that moment treatment becomes easy. The patient is allowed up and about. No dressing is required except a couple of thicknesses of gauze. The socket is irrigated daily without removing the mould. Twice a week the splint is taken down and the mould removed and cleaned. The socket is washed out and the mould and splint replaced. Slight adjustments of pressure, etc., may be required day by day.

There is no fixed time for keeping on the splint. Grafted sockets vary in their tendency to contract and in the period during which contraction is likely to continue. After three weeks a test may be made:—The clamps are removed and the rod unscrewed from the mould. If, after an hour or two, the lids show signs of gaping, the splint should be reapplied for another week. If, on the other hand, there is no attempt at extrusion, the splint may be left off until the following day, but a careful watch must be kept. The need for further splinting is decided by the course of events. It is usually necessary to retain the splint in position for three to five weeks after operation. The plaster head-cap is not removed until all risk of contraction is past. An artificial eye may then be fitted.

The operative procedure I have described is applicable to the partially contracted socket in which there is sufficient conjunctiva remaining to line the lids. The technique of post-operative splinting, however, is equally suitable for the totally obliterated socket in which all conjunctiva is lost, but a word of warning should be given here. In such a case, the remnants of the tarsal plates must be grafted and some degree of contraction and entropion will inevitably follow. If the fornices are rigidly fixed by the splinted mould, the contraction will necessarily have its effect near the lid margin and the entropion may thus be increased. This should be looked for and if signs of it appear the pressure of the splint should be relaxed a little or, if necessary, the mould should be changed for one of slightly smaller size.

The equipment required for the splint is easy to obtain. Stainless steel rod in 12 inch lengths and the universal clamps are supplied by Messrs. Down Bros. The "6 B.A." nuts may be obtained from most ironmongers. The moulds incorporating the nuts can be copied in acrylic resin from selected artificial eyes by a dental laboratory. The threaded rod to fit the moulds and the frontal plate and pin can easily be turned out by any jobbing mechanic from a length of steel rod and a small strip of sheet brass.

The moulds and the rest of the splint can, of course, be used repeatedly.

Summary

1. The advisability of conserving healthy conjunctiva in a partially contracted socket is discussed.
2. Details of operation and a method of post-operative splinting of the mould are described.

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-

ANNOTATION

The Milking-side Eye

The milking-side eye of a cow is the right side. One not infrequently finds that people refer to sides of the body in terms connected with their daily work. The Admiral who complained of pain in a tooth on the port side and Commodore Trunnion, whose larboard eye was missing, are examples from the seafaring point of view. All those who were born and bred in country districts must have visited the cow house to see milking in progress during their childhood days. That, and visits to the forge, were among the diversions of our own youth. One remembers that the milker sat on a three legged stool on the right hand side of the animal with the cheek alongside the flank of the cow and face directed towards the caudal end of the beast. If milkers sat in any other position the cow would be unable to see what was going on, might get restive and upset the pail. The importance of this in these austerity days can hardly be overestimated.

A correspondent recently wrote to tell us of a farmer who had asked him to have a look at one of his heifers which had something wrong with its milking-side eye, and we heard later that the owner had developed a conjunctivitis in his own right eye. It may be recalled that Angel Clare chose the milking time to declare his affection for Tess. "Old Pretty by this time had looked round, puzzled; and seeing two people crouching under her, where, by immemorial custom, there should have been only one, lifted her hind leg crossly."

In view of the mechanical aids practised in milking at the present

time we shall not be surprised, if in a few year's time, very few hand milkers will be left in this country. Handicraft is being superseded by mechanism; in our own opinion to the great loss of the amenities of village life. Few youngsters are taught nowadays to use a scythe and the same will soon be true of thatching. In the farm yard a Dutch barn solves this question and they were being used when we were a boy. But, with prefabricated houses, the old-fashioned thatched cottage will not last much longer.

The High Church parson who advertised in a local paper for "a communicant who can milk" could not have envisaged the time when it would be impossible to obtain the services of a milk-maid, whether a communicant or not. It seems worth while to rescue the milking-side eye from oblivion before it is too late.

CORRESPONDENCE

COLOUR VISION IN THE CONSULTING ROOM

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—It is not easy to understand why Dr. Grieve still wishes to compare the percentage of colour defectives in my series of cases with the average found in the whole male sex. If the figures had been nearer to one another it would have pointed to a serious defect for, as I have stated, my cases were not chosen at random and some groups had been combed for defectives before coming to me. To criticise the low figure after my explanation is quite illogical.

I attempted to follow precisely the printed instructions when using the Ishihara plates, the Pseudo-Isochromatic plates, the Giles-Archer Colour Perception Unit, and the Edridge-Green Lantern, for details of which Dr. Grieve can peruse the relevant booklets. I have described the method of using my own lantern.

Dr. Grieve knows that for some years many of us were unable to avail ourselves of laboratory facilities, and investigations had to be carried out under adverse conditions. He must realise that it was quite impossible to have laboratory-calibrated scientific instruments built to special design, and improvisations had to be used. Now that these obstacles are removed the findings can be investigated in the laboratory, but 40,000 cases cannot be examined "when seated in an arm-chair by the fire"; conclusions drawn from small numbers of cases are inconclusive.

He is right when he states "the efficient testing of colour vision is no easy task." When I found some men who had been considered "normal," "safe," and "unsafe" by different specialists, I realised this, and conducted the investigation in an attempt to find improvements in the efficiency of the methods of testing, and in the apparatus used.

Yours sincerely,

FRANK R. NEUBERT.

HAUTERIVE,
THE QUEEN'S ROAD,
GUERNSEY.
October 10, 1947.

OBITUARY

G. W. THOMPSON

GEORGE WILLIAM THOMPSON died recently at the age of 82 years in Argentina. His medical education was obtained at Edinburgh University where he qualified in 1890. He became a Fellow of the Royal College of Surgeons of England in 1901.

When I knew him between 1900 and 1903 he was Chief Clinical Assistant to William Lang at Moorfields, and a very pleasant and unassuming fellow he was. He was Lang's Chief Clinical Assistant from 1898 to 1905.

His appointments included those of Surgeon to the Western Ophthalmic Hospital, and Ophthalmic Surgeon to the French Hospital and to the Maida Vale Hospital for Nervous Diseases. He was a Member of the Ophthalmological Society, U.K., from 1905 to 1930.

He retired from practice some years ago. His address in the current number of the Medical Register is given as 11, Upper Wimpole Street. He paid me a friendly visit eight or nine years ago when I understood that he had interests in Argentina. I regret his loss.

A. F. MACCALLAN.

NOTES

Appointment MR. PHILIP M. WOOD, M.B., Ch.B., D.O.M.S.,
has been appointed Visiting Ophthalmic
Surgeon to the Royal Halifax Infirmary.

* * * *

**The Eye-Bank for
Sight Restoration, Inc.
New York City** NEW YORK CITY The Ophthalmic
Society of New Zealand has recently organized
an "eye-bank scheme for corneal graft
material," according to word received at national headquarters of
The Eye-Bank for Sight Restoration, Inc., 210 East 64th Street,
New York City.

Dr. Cecil Pittar, an ophthalmologist in Auckland, New Zealand, in a letter asking for advice and assistance. describes the clearing house for corneas established by the Ophthalmic Society in his country. Dr. Pittar, who is acting as registrar for the new project, is notified by all hospitals and ophthalmic surgeons whenever it is planned to enucleate an eye with a good cornea. He is also notified by any surgeon who is in need of corneal graft material. Thus, he is able to arrange for the cornea to be transplanted within 24 hours.

It is planned to send enucleated eyes by air from one part of the country to another, Dr. Pittar writes. Plans are being made, also, to have "a pathological examination of the enucleated eye at the recipient end or to have the remains of the eye returned for pathological examination at the centre of the enucleation."

In response to Dr. Pittar's request, the New York Eye-Bank for Sight Restoration, Inc. has forwarded to him samples of the bottles and containers which are used for transporting eyes in the United States.

* * * *

**The
Ophthalmological
Society of
Australia** THE Ophthalmological Society of Australia
proposes to hold its Eighth Annual General
Meeting at Perth, Western Australia, on
August 15-21, 1948, in conjunction with the
Sixth Session of Congress of the British Medical Association in
Australia.

The President of the Section of Ophthalmology of Congress is Dr. J. Bruce Hamilton, 174, Macquarie Street, Hobart, Tasmania, and the Honorary Secretary, Dr. John L. Day, St. George's Terrace, Perth, Western Australia.

Overseas visitors are cordially invited.

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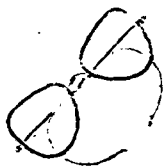
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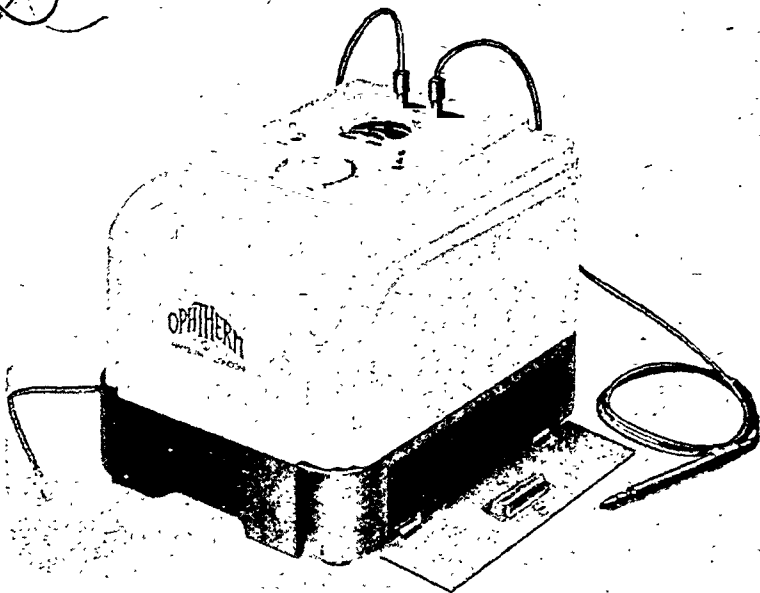
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THE BRITISH JOURNAL OF OPHTHALMOLOGY

DECEMBER, 1947

COMMUNICATIONS

A CASE OF METASTATIC OSTEOSARCOMA IN THE CHOROID*

BY

V. T. LEES

BLACKPOOL

THE occurrence of metastatic sarcoma in the eye is an event of such rarity, to judge from the few cases reported in ophthalmic literature, as to indicate the publication of a case which came to my attention whilst on active service recently.

To the best of my belief this case is the only recorded instance of an osteosarcoma in the eye.

In 1888 Meigs and de Schweinitz described a case with metastases in the 2nd and 3rd nerves in the cranium, and in the 2nd nerve and in the muscles within the orbit. Both eyes were affected by deposits in the choroid.

The primary growth was a round cell sarcoma in the chest. The spread of metastases was presumed to be from an erosion of the carotid artery found at post-mortem examination.

* Received for publication, May 29, 1947.

A similar case was found by Weiner and described in 1902, and yet another by Ballantyne in 1905, in which the primary sarcoma was in the chest and caused metastases in the suprarenal gland and brain, with deposits in the optic nerve spreading from the disc to the retina.

Heine in 1899 described a metastatic sarcoma confined to the optic papilla.

Schiess Gemuseus and Roth in 1879 attributed a pigmented tumour of the disc to spread from a malignant melanoma of the skin.

In 1907 Neese reported a choroidal tumour derived from sarcoma of the breast, and more recently Elschmig, 1926, reported a tumour of the iris of spindle cell type originating from a primary lesion of the ovary.

The case which I now describe, starting as it did with a sarcoma of the knee, has the characteristic bone cytology so well marked in the ocular deposit as to prove its origin without any doubt.

Another interesting feature in this case was that it was possible to examine the tumour *in vivo* by means of the slit-lamp microscope, though with the magnification available no point of special importance was brought to view by this means.

I saw the patient in May, 1946, at No. 19 General Hospital in the Middle East.

About one year previously the patient injured his knee and developed a sarcoma at the site of injury.

In spite of amputation of the leg, metastases appeared in his chest later in the year.

Two months before consulting me, the patient noticed a vague visual defect in his right eye, which progressed to almost complete loss of vision by the time his eye was examined.

On examination a patch of scleral hyperaemia was present in the lower nasal area.

The anterior chamber was shallow, the pupil dilated and unresponsive to direct light but reacting consensually with the opposite eye. Ocular movement was full and free.

The fundus showed a detachment of the retina present, separable into two parts contrasting in appearance, and caused by a tumour with serous detachments above and below.

The tumour was double, both parts being in the lower nasal quadrant and appearing to be the result of an embolus of tumour cells lodging possibly in a ciliary vessel.

One nodule extended from the disc to about the equator and the other from the equator forwards. In colour they were pink with rounded hemispherical surfaces elevating the retina.

The anterior nodule was clearly seen with the slit-lamp and microscope and appeared to have a loosely fibrillar structure on the surface. No pigmentation was visible.

Transillumination clearly demonstrated the solid nature of the nodules.

Examination showed nothing abnormal in the patient's left eye, nor was any C.N.S. lesion demonstrable.

The patient experienced no pain during the period of observation.

Later the detachment became total and the tumour not visible as a result.

Enucleation was not contemplated as there was no pain to justify such procedure.

Owing to my return home from active service I was unable to follow up the case, but Lt.-Col. J. Anderson, R.A.M.C., kindly obtained post-mortem sections of the eye for me.

These have been examined by Prof. Baker of Manchester University Pathology Department who reports:

"The sections show an osteo-sarcoma forming irregular trabeculae of osteoid tissue. The centres of a few of these trabeculae show calcification, but the bulk of the tissue is non-calcified. The tumour cells show rounded or oval nuclei, and there are some irregular larger types showing the cytology of malignancy. The tumour is a sarcoma of bone origin."

We have therefore in this case an indisputable chain of evidence to prove the diagnosis.

It commences with a known source of bone sarcoma in the patient's knee, which spreads to the mediastinum, and from the



FIG. 1. X20

mediastinum to the eye, probably by vessel erosion such as was found by Meigs and de Schweinitz in their case of metastatic intra-ocular sarcoma.

The occurrence of a double tumour in the eye indicates firmly that this growth is of metastatic origin, and pathological examination of sections of the ocular deposits shows such a distinctive cell pattern, that from this alone it is possible to diagnose bone sarcoma without reference to the previous history.

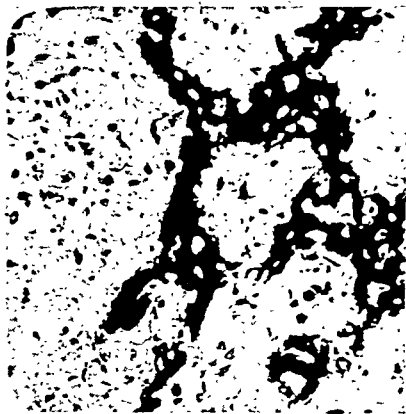


FIG. 2. X200

Reproductions of photomicrographs of the ocular tumour are published herewith, and show the trabeculae of osteoid tissue with the tumour cells in the interstices.

The dense black areas represent the calcified portions of the tumour.

The sections were stained with haematoxylin and eosin.

I wish to thank Professor Baker for his kindness in examining and reporting upon the tumour sections and for supplying the photomicrographs. I also desire to express my appreciation to Lt.-Col. J. Anderson, R.A.M.C., for his invaluable help in securing the sections of the tumour and forwarding these to me from the Middle East.

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THE EFFECTS OF NITROGEN MUSTARD ON THE PERMEABILITY OF THE BLOOD-AQUEOUS HUMOUR BARRIER TO EVANS BLUE

An instance of the influence of a lesion in one eye on
the susceptibility of the other*

BY

HUGH DAVSON and J. P. QUILLIAM

PORTON AND LONDON

THE chemical warfare agent, "nitrogen mustard" or methyl bis (β -chloroethyl) amine, has come to the notice of the clinician lately on account of its beneficial effect upon Hodgkin's disease, the leucaemias and some neoplasms (Gilman and Phillips, 1946). It is also of interest to those studying the problems associated with the changes in the permeability in the blood-aqueous humour barrier since exposure of the eye to nitrogen mustard either in the liquid or vapour form results in a well marked iridocyclitic type of lesion which, in severe cases, may be associated with iris haemorrhages. Similar effects are obtained by dropping a one per cent. aqueous solution into the eye.

The membranes separating the blood from the aqueous humour show a high degree of selectivity; thus the aqueous humour is almost completely protein-free; sucrose, injected into the blood, appears in the aqueous humour only very slowly, whilst the trisaccharide, raffinose, does not appear in the aqueous humour at all (Weld, Feindel and Davson, 1942). Similarly, a large number of dyes, when injected into the blood stream, do not appear in the aqueous humour. (Halpert, German and Hopper, 1933, *Amer. Jl. Physiol.*, Vol. CIII, p. 351). It was considered possible that the damage in the uveal tract caused by nitrogen mustard would manifest itself in the form of an increased permeability of the membranes separating the aqueous humour from the blood stream. If this were indeed the case, then it might be expected that a dye which, when injected into the blood of the normal animal, failed to appear in the aqueous humour, would appear in the aqueous humour of an animal whose eyes had been exposed to this chemical agent. Preliminary experiments showed this to be true; thus chlorazole fast pink, when injected into the blood of the normal rabbit, failed to appear in the aqueous humour; however, after exposure of the animal to nitrogen mustard vapour, the aqueous humour was found to be pink, indicating a change in the selective power of the membranes. The dye finally chosen for these experiments was

* From the Chemical Defence Experimental Station, Porton, and the Departments of Physiology, University College and King's College, London.

Evans blue 1024 which has the advantages of being eliminated very slowly indeed from the blood stream, and of giving an intense blue colour at low dilutions.

The work to be described here was undertaken to find out whether the rate of penetration of Evans blue into the eye could be used as an index to the severity of a lesion caused by nitrogen mustard and to this end the effects of three different preparations of the substance, known to have different toxicities, were compared. The results showed that this was indeed possible; moreover an extremely interesting dependence of the severity of the lesion in one eye or the condition of the other eye became manifest and for this reason that part of the work bearing on this point is being published.

Experimental

Aqueous solutions of nitrogen mustard were used; on dissolution in water the reagent undergoes a series of changes with time, its activity rapidly diminishing and then rising to a constant value. Thus there were three solutions which could be used, namely nitrogen mustard dissolved in ice-cold water and immediately applied to the eye; the same solution kept for 10 minutes at room temperature, in which case the effectivity would be expected to be minimal; and finally the same solution kept for five hours at room temperature. Toxicity studies had indicated that the order of effectiveness would be:—

A	B	C
Ice-cold	5 hours	10-minutes

Rabbits were injected with 1 ml. of a 1 per cent. solution of Evans blue 1024 in isotonic saline per 5 lb. body weight. 60 mg of the appropriate solution were instilled into the two eyes. Two hours after the instillation, the aqueous humours were removed from both eyes and the concentrations of Evans blue determined colorimetrically. The severity of the lesion was assessed in terms of the dye concentration in accordance with a somewhat arbitrary scale, the maximal possible dye concentration (namely that in the blood) being given the value 100, and lower concentrations values in proportion. If we wish to compare the lesions produced by the two weaker solutions B and C, we may proceed in two ways:—

(a) A standard lesion may be produced in one eye of all animals with solution A; in one group the other eye would have solution B instilled into it, and in the other group the other eye would have solution C. The ratio: Lesion A/Lesion B could be compared with the ratio: Lesion A/Lesion C.

(b) Alternatively, in a group of rabbits, solution B could be

EFFECTS OF NITROGEN MUSTARD ON THE PERMEABILITY OF THE BLOOD-AQUEOUS HUMOUR BARRIER TO EVANS BLUE 719

instilled into one eye and solution C into the other, and the lesions directly compared.

If the two eyes of an animal can be treated as independent, we may expect to obtain the same results with both procedures.

Results

When procedure (a) was used solutions B and C produced lesions of intensities of 17 and of 11 respectively (Table 1), solution B being the stronger as toxicity studies indicated. When procedure (b) was adopted, solutions B and C being instilled into the two eyes of the same animal, the lesions were (Table 2) 12 for solution B and 1·6 for solution C, i.e., solution B was certainly stronger in effect than

TABLE 1

The lesions produced by three preparations of nitrogen mustard (Solutions A, B and C). In all animals, solution A was instilled into the left eye. The intensity of the Evans blue in the blood was taken as 100 and the values in the aqueous humours recorded below were in proportion and were estimated colorimetrically.

No. of Rabbits	Average Value of Lesion produced		
	L. Eye	R. Eye	$r = \text{L. Eye/R. Eye}$
18	(Solution A) 35	(Solution B) 17	2·3
22	(Solution A) 36	(Solution C) 11	3·6

TABLE II

The lesions produced by the two weaker preparations of nitrogen mustard (Solutions B and C). In all animals solution B was instilled into the left eye, solution C into the right.

Method of estimation as referred to in Table 1.

No. of Rabbits	Lesion		
	L. Eye	R. Eye	$r = \text{L. Eye/R. Eye}$
18	(Solution B) 12	(Solution C) 1·6	9

solution C, as indicated in the first series; but with this procedure it appears to be some nine times as effective whereas with procedure (a) it appears to be only about one and a half times as effective. The reason for this difference, as the Tables show, is that the weakest preparation, solution C, produces a very small lesion (1.6) when the other eye has solution B in it, whereas it produces a considerably larger one (11) when solution A is in the other eye. Solution B produces a larger lesion when solution A is in the other eye but the effect is not so great, so that the effect of passing from the one procedure to the other is to modify the ratio: Lesion B/Lesion C very considerably.

Now the solution A is the most effective in causing lesions (it causes a lesion of 36 when solution C causes 11, and 35 when solution B causes 17) and the results can best be explained if the instillation of the powerful solution A tends to produce an incipient lesion in the other eye; the effect of the weakest solution (C) will therefore be stronger when solution A is in the other eye than when solution B has been instilled.

Discussion

The two eyes of a rabbit are not necessarily to be viewed as mutually independent organs but rather each as being individually susceptible to changes going on in the other. The reciprocal action is probably reflex in origin, since it is hard to believe that the small quantities of toxic material actually absorbed into the blood from the conjunctival sac could be sufficient to have any general effects by way of the blood stream. The effective barrier which separates the anterior chamber from the blood in the anterior part of the iris is possibly only that due to the endothelial lining of the capillaries, since the endothelium of the iris contains lacunae of sufficient size to prevent it from acting as an intact membrane. It is well known that the permeability of the capillary endothelium to proteins, and therefore to Evans blue, is largely determined by the physiological state of the capillaries; if they are dilated their permeability is high, if contracted, it is low. If the lesion produced by a high concentration of nitrogen mustard in one eye causes a reflex dilatation of the vessels in the iris of the other, then the phenomena described here are explicable on the basis of reflex action. In confirmation of this viewpoint it has been noted that by instilling nitrogen mustard into the left eyes of a group of animals a ciliary injection was observed in the untreated right eyes; only on one occasion, however, was it possible to demonstrate that injected Evans blue appeared in the contra-lateral eye. We may therefore conclude that a severe lesion in one eye potentiates the action of a mild nitrogen-mustard agent in the other by reflex action; in the present case the effects of this

potentiation are greater with the milder solution C than with the stronger solution B.

We are indebted to Sir Stewart Duke-Elder for advice and to the Chief Scientist, the Ministry of Supply, for permission to publish this paper.

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PERIPHERAL AND CENTRAL DISTURBANCES OF THE VISUAL FIELDS. AN ASPECT OF DI-OPHTHALMOLOGY*

BY

N. A. STUTTERHEIM

JOHANNESBERG

THE cause of eyestrain, concomitant squint, and amblyopia used to be unknown, the diagnosis incorrect, the therapy inadequate and therefore the prognosis dubious. It was not until convergence had been recognized as the kinetic principle of bi-foveal vision^{1,2,3,4} that the dynamic factor of human binocular vision was established. From then on the diagnosis, prognosis as well as therapy of these disorders could be shaped on rational lines within the precincts of physiology where they belong. These disorders concern the physiology of the bi-foveal eye, the di-opthalmos, of which the bi-fovea is the highest achievement.

If each of the three following cases were assessed by the usual standards of ophthalmology, they would have a very sombre prognosis. In these cases, there was no workable diagnosis and therefore no therapy, until they could be recognized as disorders of the di-opthalmos.

Case I

L., a schoolboy, aged 17 years, was referred to me by his uncle, a specialist-physician; on July 7, 1946 (abbreviated letter):—

I have referred L. du P. to you. His parents are very concerned as to his condition, and would like to know what the future holds.

* Received for publication, March 25, 1947.

Apparently he has had a limited field of vision all his life. At school, less than a year ago, when the eyes were called upon to do very minute work, he had an attack of blindness followed by severe headache, and then an ophthalmic surgeon discovered the real condition of affairs.

What the parents now are anxious to know is:

- (1) What is precisely the nature of his complaint?
- (2) What is the prognosis—is total blindness to be expected, and within what period?
- (3) Will any treatment help or retard the condition?
- (4) Do you advise that he be sent to an Institute for the Blind?

If you think it necessary that a neurologist should also see him, then you are at liberty to make the required arrangements.

L. was brought by his sister because he could not cross the streets by himself. He entered with hands stretched sideways as if feeling for doorposts.

Patient was depressed in his mind. His only desire was not to be examined any more but to go to a school for the blind and learn Braille so as to be able to read and also to learn a trade. He could read printed matter letter by letter only and a page took him "a whole day." From his first school year onwards he saw as though through a funnel, with both eyes together and with each eye separately. The reason why he had never complained—and so his deficiency had not been discovered earlier—was that he knew no better but that such was the case with everybody else. He frequently had headaches, often severe ones, sometimes causing him to faint.

Drawing and reading he could not do without strain and headache nor could he go to film shows. He always became giddy when looking out from a moving train. There was no night-blindness and he could orientate himself well when moving about in his home surroundings, even better at night than in daylight.

He could not play the piano with both hands because he could not see them at the same time. Since his tenth year he noticed limitations of his temporal visual fields. This became worse. When thirteen years of age he had an attack of blindness which lasted about an hour, followed by severe headache for the whole day, and he had to retire to bed. He experienced no nausea or giddiness on that occasion.

Similar attacks occurred on four occasions, the last and worst attack being in March, 1946, during a drawing lesson. He had started seeing spots within the narrow funnel of his vision. He then was sent to an ophthalmic surgeon, and afterwards his uncle

also examined him. The ophthalmic surgeon sent him to a radiologist and to another specialist-physician. After March he left school altogether.

The boy had difficulty in finding the next letter in each row of Snellen's type, but slowly he could distinguish all the letters of the 6/12 row with each eye.

His fields, taken with a white square of 1 cm., were similar in the right and left eye. They did not extend beyond the 11 degree circle and in some meridians only had 9—7 degrees from the centre-point, but in a circle at 25—30 degrees distance from the centre-point, flashes of the moving white square were observed—with each eye.

A neuro-psychopathic disposition was excluded (*vide infra*). Patient was a strong, healthy, intelligent boy. His uncle confirmed this impression and corroborated L.'s story. He further wrote:—

The ophthalmic surgeon consulted (in March, 1916) gave a very unfavourable prognosis and, I understand, expressed the opinion that L. was soon to go totally blind.

In July (1916) his mother wrote to me that L. was steadily getting worse and that he himself was very worried that he soon would be blind, and that she had arranged for his admission into the Institute for the Blind at Worcester, and that she had received his railway concession to go there.

After this the uncle persuaded the parents to send L. to Johannesburg for consultation.

The report on the X-Ray examination read: The examination revealed no enlargement of the pituitary fossa and there was no evidence of increased intra-cranial pressure. The optic foramina were normal.

The second specialist-physician could find nothing positive, and had written accordingly.

It is evident that ophthalmologically nothing had been neglected in order to arrive at a diagnosis, and also that the ophthalmic surgeon was justified in warning the parents as to the seriousness of the condition, as orthodox ophthalmology could give no clue to the real condition. His prognosis was bad: blindness of both eyes, already far advanced, was increasing and, evidently, total blindness impending.

The psychiatrist to whom L. had been referred by me, wrote:—

"I can find nothing that can throw light on the cause of his condition. There is no question of any organic lesion or neurological deviation. Psychologically this patient is perhaps a little tense, but I cannot find any neurotic or psychotic symptoms, and there decidedly is no sign of any inclination to hysteria. I have therefore a perfectly negative report to render.

I should be pleased to hear the further progress of this interesting case."

In view of the extraordinary features of the case and especially because of the bad prognosis, I asked the opinion of two other ophthalmologists. I showed the letter of introduction by L.'s uncle, the specialist-physician. I refrained from telling them my diagnosis and also that treatment had already been instituted on the strength of that diagnosis. This may explain the slight difference in their and my assessment of visual fields and acuity. He was already more self-confident, as can be seen in the report by one of the two ophthalmic surgeons. It reads as follows:—

Herewith report on L. du P. (July 29, 1946). Examination revealed a normal fundus each side, with visual acuity 6/9 in each eye, with negligible refractive errors. He walked about the place without difficulty, totally unlike a person who had a visual field contraction as is shown on the visual field charts, which I enclose. The visual fields were contracted to 10 degrees (ranging in places to 20 degrees) around the centre-point, with a slight paracentral ring scotoma (relative) within the existing field. Neuro-ophthalmologically I could establish nothing else. Having suspicion that this was a case similar to the case described in your book, I advised him to undergo your treatment.

The other ophthalmic surgeon informed me verbally that he could establish no organic cause for the condition and that it was a case of functional field contraction. His field charts were similar to those of the other doctor and myself. Obviously no adequate diagnosis was made and consequently no rational therapy proposed.

The further progress was that within ten days visual acuity had become normal (6/6) in each eye and that within three weeks after the first, a further report by the one consultant, who had kept in touch with the case, reads:—

Examination on August 13, 1946, showed marked improvement in the fields, there being an almost full field, filled in by relative scotoma extending from the periphery up to the region of 25 degrees on an average.

A fortnight later a third report reads:—

Examination on August 29, 1946. Normal fields with small irregular scotomata about 10 degrees from centre-point, O.D.S.

This is a case of similar type to the one described by you, viz., functional peripheral amblyopia. I am sure this cannot be called a hysterically contracted field, and if so, then you have a very effective treatment to offer. If not, which I think is the case, then you have discovered a new condition and its treatment.

In conservative ophthalmology the prognosis of the case of L.

du P. was bad. Conversely my prognosis was good, because the diagnosis is:—

Functional contraction of the visual field of each eye and diminished central visual acuity (amblyopia), combined with various other symptoms of severe eyestrain, caused by asthenovergence.

Initially convergence was low, ranging from "negative" convergence: minus 5 prism degrees to (positive) convergence: 6 prism degrees; a range, therefore of only eleven degrees of prism (instead of ninety degrees of prism, which can be considered as normal range of reflex-convergence).*

In the first ten sittings of kinetic treatment for asthenovergence it was rather difficult, technically, to go beyond that range upwards (i.e., to increase the "positive" side of the range) because of the contracted field in each eye, as the second image disappeared too readily in the scotomatous area. Treatment was given twice daily for about ten minutes each time.

Progress was rapid. After thirty-eight sittings of kinetic treatment, the range of convergence was from minus 5 to 21, the fields and visual acuity were normal and depression of spirit had made place for energy. In six weeks' time L. could read fluently. That week and the next, by his own choice, he read a complete popular-scientific book (164 pages). He had himself registered for a two months' course of drawing and modelling, and produced good work there.

Three months after he began treatment he was back at school, making up for lost time.

Altogether eighty-seven sittings of kinetic treatment for asthenovergence had been given during two-and-half months; no other treatment.

Case II

Another case exemplifying a good prognosis in the light of our knowledge of di-opththalmology is already on record. It has been described by me in 1937, in the monograph, "Eyestrain and Convergence," pp. 38-41. As, however, this description has apparently passed unnoticed, no mention being made of it, critically, in reviews of the book in the medical press, I feel justified in quoting that chapter (abbreviated).

The title is:—

"Eyestrain with Field Contraction."

"Another visual symptom of eyestrain that deserves special

* For definitions and the meaning of some terms in di-opththalmology, see my monographs "Eyestrain and Convergence," pp. 1-16, and "Squint and Convergence, a Study in Di-opththalmology," pp. 5-9.

attention is contraction of the visual fields. This disorder in the wake of eyestrain is probably not so rare as our limited experience would seem to suggest. The following case shows that it can assume serious proportions."

In July, 1927, G., a farmer's son, aged twenty-one years, consulted me complaining of frequent headaches and other symptoms of eyestrain of long standing, but his main complaint was some cloudiness in front of his left eye.

Refraction of each eye was normal: a mixed astigmatism of 0.5 dioptre with vertical axis in each eye. Vision of right eye 6/4, of left eye 6/9, with correction (-0.75 D.sph.) 6/4. The fields, as far as a cursory examination revealed, were normal. Ophthalmoscopically or otherwise I could find nothing to account for patient's complaint, except astheno-vergence. Convergence was 12 degrees of prism, negative convergence -5 degrees.

G. could not stay for further observation or for treatment, but I saw him again four years afterwards.

In April, 1931, I was again consulted by G., now twenty-five years of age, a farmer of healthy intelligent appearance. He looked tired and complained that for sixteen months now his left eye gave him the impression of looking through a narrow tube, narrower in full light than in the shade or dark. Since boyhood he had suffered from headaches, occasional giddiness and nausea. He experienced discomfort when reading and had largely abandoned it. Moving pictures irritated him and he had given up going to film shows. He wore tinted glasses, slightly cylindrical. They gave relief against glare but made no difference as to his eyestrain. His right eye saw well.

During the period since his former consultation of me, G. had been regularly attended to ophthalmologically elsewhere. Now his general medical attendant wrote to me that: he complains of headaches and failure of vision. . . . His discs look normal and I find no cause for his failure of vision. . . . Beyond testing the peripheral range of vision and finding that it was very deficient, especially in one eye. . . .

These quotations from his family doctor's letter show that this was not a neglected case, but that it had received apart from the specialist's help the able and careful attention of the local practitioner.

G. was very concerned about the extreme narrowing of the field of his left eye, stating that sometimes at street corners he bumped into people coming from the left. He was afraid of losing the sight of that eye altogether.

Vision of right eye 6/4 (uncorrected), of left eye 6/9 (uncorrected). Glasses made no improvement. It was difficult to find

the next letter in the row with his left eye, the right being covered.
 Refraction: mixed astigmatism 0.5 with vertical axis for each eye.

Perimetric examination with white object 1 cm. square. Right visual field showed concentric contraction; in the temporal meridian it was only 75 degrees. Left visual field was 5 degrees wide in every direction, measured with white object 1 cm. square in ordinary daylight, on the Bjerrum screen.

Convergence was 12 degrees of prism, negative convergence 2-5 degrees.

Ophthalmoscopic examination of media and fundus of each eye was negative. An organic cause for the contraction of the visual fields could not be found, and toxic causes not traced in the history. Blinding by snow or other strong light was out of the question. Consequently, it appeared to be a case of functional contraction of the visual fields.

There was no evidence in the history or present state of the patient which could point to a psycho-neurotic condition.

In the text-books three conditions only were mentioned as causing functional contraction, viz., hysteria, neurasthenia and traumatic neurosis.

The last-mentioned was excluded, as there was no trauma in the history of G.'s case; moreover, as an independent farmer he was not likely to develop that disorder.

Neurasthenia can be accompanied by *moderate* contraction only, a sign of abnormally quick exhaustion (Oppenheim's). Evidently that did not apply to G.'s case of extreme contraction.

Consequently, hysteria only was left for further consideration. The neurologist who, on my request, examined G., reported:—

I was unable to elicit the presence of any symptoms which could be definitely labelled as psycho-neurotic. On examination he presented no abnormal signs except a certain degree of hyposensitiveness of the palate and soles. As this condition is present in a large proportion of normal people, I do not think that it assists in the diagnosis.

Hysteria, therefore, could be excluded, the more so as functional contraction of the visual field in a case of hysteria always is a sign of a severe form of the disease. Jelgerma writes in this connection⁶ (translated):

"In hysteria various disorders of the visual organ occur rather frequently. They always are evidence of a rather severe form of hysteria. Concentric contraction of the visual field is the best known symptom and diagnostically of the greatest importance . . . so that the patient in some cases has merely left a few degrees round the fovea centralis with conscious perception of visual

impressions. *This form of contraction always is a striking revelation of severe hysteria . . .* Striking features are . . . the lack of symptoms produced. *The patients themselves know nothing of it, and it cannot be noticed in their daily lives or in their movements and actions."*

The contrast of this description with the case of G. is obvious. There could not be a shadow of doubt that G.'s case was not one of hysteria.

I think it necessary to stress this point because to my knowledge the diagnosis of extreme contraction of the visual fields as a sequel of eyestrain has never been made before. I wish, therefore, to make quite clear the complete exclusion of any alternative diagnosis in this case.

The history shows that this was a case of severe eyestrain of long standing. I knew, by my examinations of G. in 1927 and 1931, that he had suffered from uncorrected astheno-vergence for many years. I had measured the convergence and had found it very low on both occasions.

The only course open, therefore, was to treat the case according to the following diagnosis:

Functional contraction of the visual fields of both eyes and diminished central vision of the left eye (amblyopia), combined with various other symptoms of severe eyestrain, caused by astheno-vergence.

The treatment was kinetic.

The ensuing improvement during forty-three daily sittings of kinetic treatment (of ten minutes each) was a model of steadiness. The extremely contracted left field opened up very slowly at first but regularly day by day, until after thirty-six sittings the field was normal. The right eye's field lost its contraction during the same period. Central vision of the left eye increased in acuity, without the help of glasses, from 6/9 to 6/4. Headaches disappeared. G. took to reading again without discomfort. Photophobia was the last symptom to disappear, and G. discarded his dark glasses.

This prompt and complete cure has now lasted more than fifteen years. There never was any recurrence of symptoms.

There are probably many cases of eyestrain connected with functional contraction of the visual fields. In the May number, 1930, of the *American Journal of Ophthalmology*, Rowe and Rowland draw attention to the fact that in a general medical service amongst 1,900 patients, 100 were found to have a marked concentric contraction of the visual fields. In half this number the condition could not be attributed to commonly recognized causes. The paper closes with the remark: "There are

apparently many cases of marked field contraction in which the condition has not been detected."

I suggest that in many such cases the cause may be asthenovergence.

Discussion

The two cases of field contraction described in this paper can be considered as extreme cases representative of a large group. My subject, however, is not what they are representative of, but that they are typical forms of field contraction for which there is yet no name and therefore no apparent therapy. In the literature no mention is made of this form of field contraction (except once by me). It probably becomes grouped, unwittingly and wrongly, with other groups of functional field contraction for which there is given an accurate diagnosis, but *the kind of field contraction under consideration here has not yet been recognized as a clinical entity*. Therefore there exists no therapy, and as the condition is progressive diminution of sight, the prognosis must appear bad. This is what the practical ophthalmologist is faced with in cases of this type, unless he realizes its meaning in di-opththalmology. As soon as the condition is recognized as a bi-foveal matter, showing its main disturbances in the visual field, then the position is clear and therapy rational and effective.

Case III

Another typical case, this time not of peripheral but of central amblyopia, once more emphasizes the importance of di-opththalmology.

A girl, aged 16 years, had been prescribed -1.0D sph. for each eye. She was studying the piano but was told by the ophthalmologist to give up music and all study, as there was something very wrong with her eyes, which was progressive. The parents were alarmed, and May 30, 1945, came to me for a further opinion.

The history revealed severe headaches during the day-time, and also photophobia. Moving pictures irritated the eyes and caused bad headaches. Often the eyes were bloodshot. Vision, of late years, had become gradually worse. It was thought that the patient had become more and more myopic. She herself, dejected and upset, at first emphatically refused to undergo the proposed kinetic treatment.

I found visual acuity right eye 6/18, with correction (-1.0D. sph.) 6/12, visual acuity left eye 6/18, with correction (-1.0D. sph.) 6/12. Negligible refractive error. Convergence 16 degrees of

prism (principal angle). Negative convergence -5. As this clearly was a case of astheno-vergence causing eyestrain, kinetic treatment was instituted the next day. After twenty-one daily sittings of the treatment (about ten minutes each) the convergence ranged to 91 degrees of prism.

Visual acuity of each eye (without correction) had become 6/5. During the treatment the girl had become bright and lively and resumed her piano lessons. Three months afterwards the visual acuity of each eye was 6/4 (without correction) and her eyes caused no trouble. With my usual precautionary measures, *viz.*, a few sittings of kinetic treatment half a year after the cure and again one or two sittings a year later, the normal condition of the eyes remained stable.

Discussion

This therefore is another case representative of a great group and typical of a kind of condition which is not yet recognized in ophthalmology as a clinical entity, but is known to those who are versed in the facts of di-ophthalmology, which is based on the bi-fovea and its correlate convergence, the kinetic principle of bi-foveal vision.

Summary

These three cases represent a form of disturbance of field or central vision, or both, which if one does not grasp the importance of kinetic factors as revealed by the study of the bi-fovea and its correlate, convergence, must lead to a sombre prognosis. With kinetic treatment, however, which superficially would seem to affect adduction, abduction and bi-foveal fusion only, these cases are effectively cured both of peripheral field limitation and central visual failure. They emphasize the broadness of the conceptions aligned under the title of di-ophthalmology.

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GLAUCOMA SECONDARY TO FIBROCYSTIC DISEASE OF BONE

BY

J. W. E. CORY

BURY ST. EDMUNDS

W.G.W., a farmer aged 58 years, presented himself on December 3, 1946, complaining of loss of vision and discharge from his left eye. He had always suffered from frontal headache on the left side, and recalled as a youth it was often so severe that he had to cool his head on cold metal objects. He had a swelling over the left temporal region, which an old doctor had remarked upon some forty years ago.

On examination there appeared to be some proptosis of the left eye (3—4 mm.) and displacement outwards in the orbit and a bony



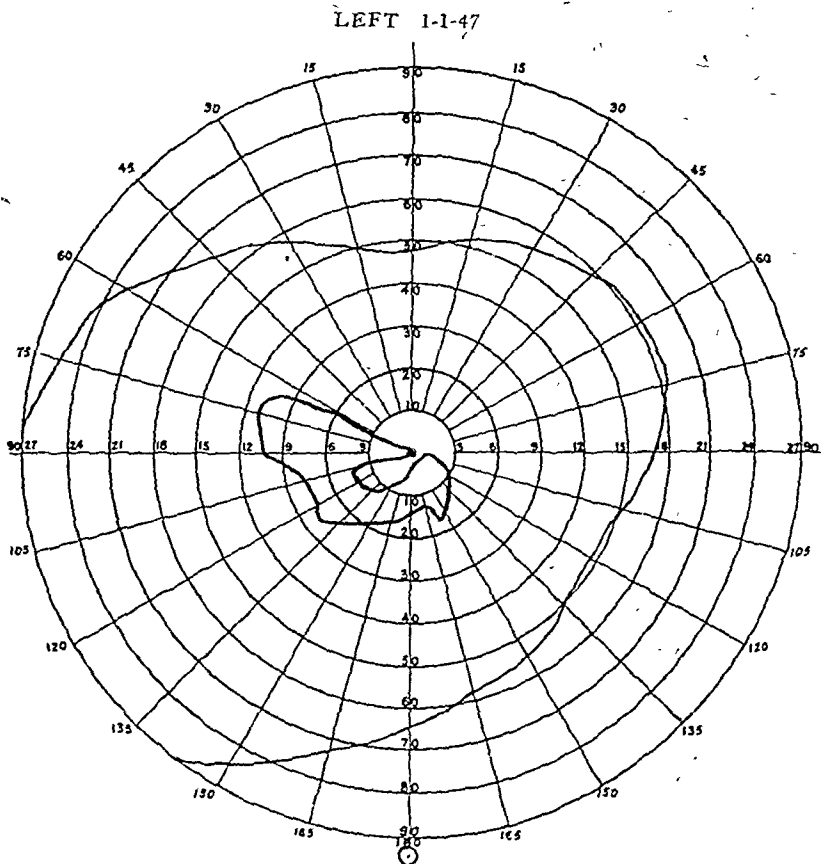
FIG. 1.

hard prominence of the fronto-temporal region on the left side. The tension of the left eye was raised. R.V. 6/9 with -0.5 cyl. 90° 6/6. L.V. 6/60 not improved by glasses. No discharge was seen and cultures from the eyes were sterile.

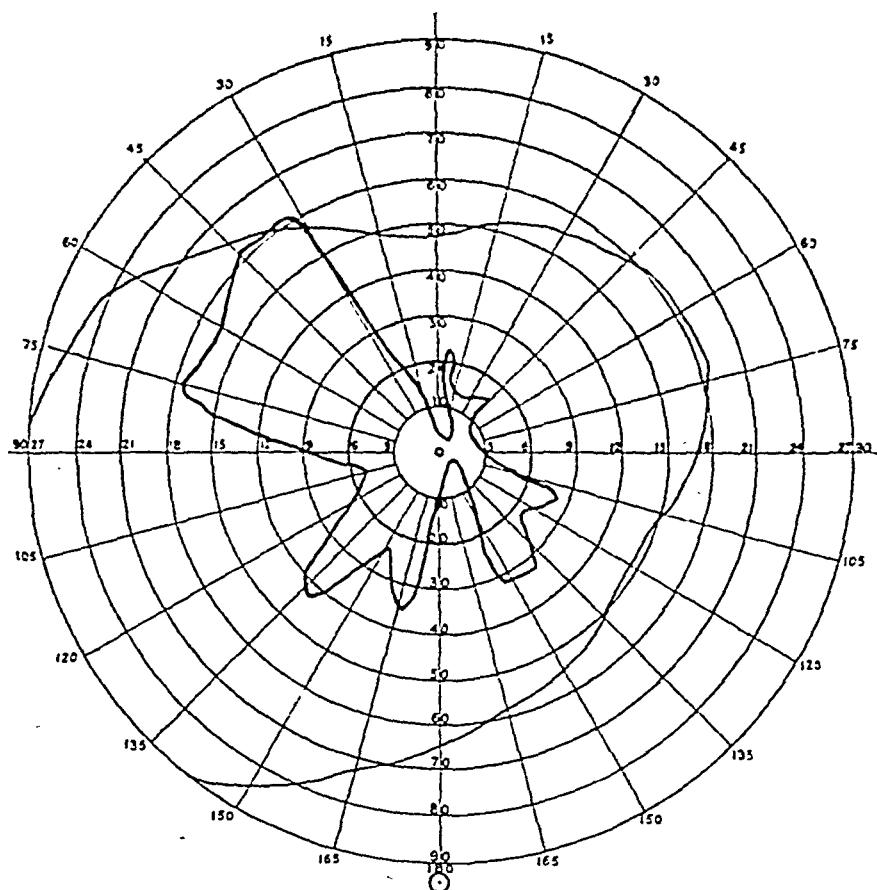
Physical examination.—There was a bony hard swelling of the left fronto-temporal region and a hard swelling over the occiput and in the left side of the jaw. The skin was normal and no fibromata were found.

His cardio-vascular system was normal, but blood pressure was 190 mm. Hg systolic and 85 mm. Hg diastolic and there was some fibrosis at the base of the left lung. Urine was normal to clinical tests.

Special investigations.—The tension of the eyes was right 25.75 mm. Hg and the left 33.75 mm. Hg average. (Schiotz tonometer with weights.) Field of vision in the right eye was full and the left restricted to a small field in the upper and outer quadrant.



LEFT 25-3-47



Ophthalmoscopy.—Right fundus was normal—the left showed a deeply cupped disc of some 6 dioptres and considerable atrophy.

Pathological Investigations.—Urine acid. Specific gravity 1022. Albumen nil. Sugar nil. Acetone nil. Centrifuged deposit nothing abnormal seen. Culture sterile. No Bence Jones proteose present.

Blood Count.—R.B.C. 5,660,000. Haemoglobin 100 per cent. Colour index 0.9. White blood corpuscles 13,400—polymorphonuclear neutrophils 73 per cent. Eosinophils 3 per cent. Lymphocytes 15 per cent. Monocytes 9 per cent. Serum calcium 9.6 mgms. per cent. Inorganic phosphate 5.0 mgms. per cent. Normal 2—3.7 mgms. per cent.

X-ray examinations showed a honeycombed condition of bone in the left frontal, temporal and occipital bones of the skull. The

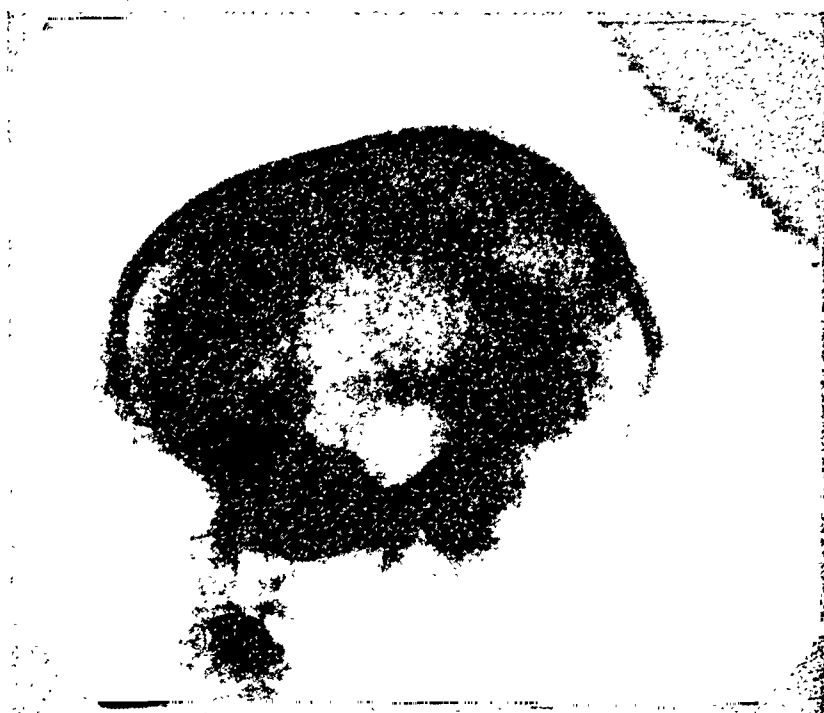


FIG 2

sphenoid was fully involved, and although the orbital fissure could be demonstrated on the right side, it could not be so demonstrated on the left side. The frontal and maxillary sinuses were obliterated on the left side although clearly seen on the right side. A complete radiological examination of the skeleton was made and the only other bones affected were the left side of the mandible and the lower third of the right humerus. Oto-rhinological examination by Mr. Ware revealed nothing of added interest.

Operation

On December 17, I performed a corneo-scleral 1.5 mm. trephine operation and his convalescence was uneventful. Subsequent tension readings were within normal limits: average 23.0 mm. Hg (Schiötz).

I think that there is little doubt that the ophthalmic condition was caused by the abnormal bony formation because it was associated with proptosis displacement and increased tension of the left eye.



FIG. 3.



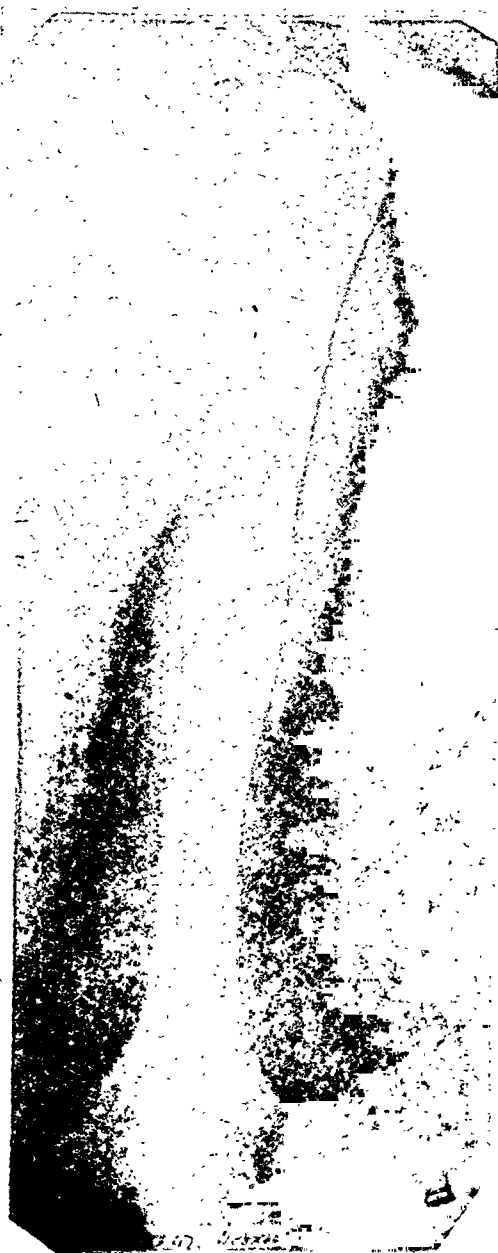


FIG. 5.

The bone condition is not a simple one to explain—at first one considered leontiasis ossium, but the fully developed condition is characterised by the leonine appearance which the name suggests and of this he has none. A malignant condition is excluded by the long history borne out by earlier photographs.

An infective condition arising from the frontal sinus in adolescence would be a feasible one, except that the condition rides rough-shod over suture lines and appears in other parts such as the left side of the mandible and the right humerus.

A diagnosis of fibrocystic disease of bone is more probable when the cystic condition of the lower end of the right humerus is examined, but until this was discovered xanthomatosis seemed a likely diagnosis to explain the unilateral involvement of the skull. The full Hans Schüller-Christian syndrome was not present.

I submitted the films to Sir Thomas Fairbank, who discussed them with Dr. Coldwell. Dr. Coldwell is inclined to consider that it is a mixture of xanthoma and fibrocystic disease, but Sir Thomas thinks that the humerus gives the key to the situation and that fibrocystic disease of the bone is the correct diagnosis. He thinks it is atypical and that there is no evidence of parathyroidism.

The ophthalmic condition suggests that the globe is displaced outwards and forwards by pressure within the orbit and that this has prevented the return venous drainage. The tension was undoubtedly raised, but it is a matter of conjecture whether the optic atrophy was the result of glaucomatous rise of tension within the eye or whether it was due to tension on the optic nerve as the result of stretching. It is true that I have been unable to demonstrate a narrowing of the orbital fissure, but the fact that it is demonstrated on the right and cannot be demonstrated on the left suggests that the fibrocystic disease of the sphenoid has so narrowed the fissure that it cannot be seen.

I suggest, therefore, that this is a case of diffuse fibrocystic disease of bone mainly unilateral, which has caused a secondary glaucoma of the left eye and optic atrophy. It is of double interest from the bone and from the ophthalmic viewpoint.

I am indebted to Sir Thomas Fairbank and Dr. Coldwell for their interest and help and to Mr. E. G. Recordon and Mr. H. A. Ware for their valuable suggestions and to Dr. S. D. Kilner for radiological examinations.

THE VARIATIONS IN THE SYSTEM OF THE TRUNKS OF THE POSTERIOR CILIARY ARTERIES*†

BY

T. SUDAKEVITCH

MOSCOW

AN attentive reader will notice the absence in the text-books of ophthalmology of a special section to describe the constant anastomoses of the ophthalmic artery, or, of more importance, the pathological eye-changes connected with them. This fact cannot be explained by the lack of scientific investigation of these anastomoses; they have been well-known for a long time. In 1900, Tichomirow, Professor of Anatomy, remarked that thanks to Zuckerkandle and Meyer's efforts all the variations of the route and branching of the ophthalmic artery are well known. Also this fact cannot be explained by the insignificant rôle of these anastomoses in the system of the ophthalmic artery and consequently in the life of the eye. Such a supposition is negated by the anatomical picture itself. It is sufficient to remember that the ophthalmic artery—as was proved a long time ago by prominent anatomists—sometimes starts by two trunks instead of one—the one from art. carot. interna and the other one from art. carot. externa; or only from art. carot. externa. Such a state of affairs is well shown in the illustration of B. Adachi, 1928, A. 1-3.

There is a great difference between a. carot. int. and a. carot. externa; first of all in their architecture and secondly by their relations. That is why the start of the ophthalmic artery only from the art. carot. ext. cannot be compared with its start from the art. carot. interna. This by itself is already sufficient to declare the rôle of the anastomoses to be essential and important for the functions of the eye, and we have to look for a reason why it was so neglected by ophthalmic surgeons.

The reason may be found in the peculiar opinion of the anatomists of the past century *re* the orbital vessels; an opinion inherited by ophthalmology. To show more clearly what I mean, I will quote the opinion of Meyer: "although there are wide limits for the ordinary distribution of arterial branches, nevertheless for the more important and larger ones there is a definite law. All authorities ascribe a certain constancy and independence to the central, lacrymal, supra-orbital and 2 ethmoidal arteries. Because of that they received a localisation—*sui generis*, and they got their

* Translated by N. Pines, London. † Paper read at the meeting of the Ophthalmological Society, Moscow.



ILLUSTRATION 1.

ILLUSTRATION 2.

ILLUSTRATION 3.

1. A. Ophthalm. starts from a. meningeal media (male 28 years of age, right side).
 2. A. Ophthalm. passes under the nervus opticus. A. lacrym. starts from a. menig. med. (male 38 years of age, right side).
 3. A. Ophthalm. starts by 2 equal trunks from a. carot. int. and art. carot. ext. through a meningeal media (male 48 years of age, right side).
- B. Adachi—"Das Arteriensystem der Japaner"—Kyoto, 1928.

own names. But the muscular and ciliary branches were chiefly considered summarily—jointly to a certain degree" (Tr. Meyer, "Zur Anat. der Orbitalart—Morph. Jahr. Bd. XII, Leipzig, 1887). In those words, as Meyer himself acknowledges, is expressed the general opinion of the anatomists, who reckon as most important only the central art. of the retina, and the ciliary ones only as secondary, described summarily together with the muscular ones. And naturally the question arises, what is then the rôle of the ophthalmic artery in the life of the eye—is it chiefly concentrated in man—especially in man—on the central retinal artery? The physiology of the eye tells us that it is not so. But the authorities who studied the anatomy of the ophthalmic artery and described its system evidently did not follow the principles of physiology. As a result, there is a missing link—the ciliary arteries—and in ophthalmology full use was not made of our previous knowledge of the anastomoses of the ophthalm. art. This is why we started to study afresh the system of the ophthalmic artery.

Before describing the results of our own researches we will briefly mention the known forms of variations in the system of ophthalmic artery—the scheme of Prof. Tichomiroff—and adding to it some illustrations (M. A. Tichomiroff, "Variations of the

arteries and veins of the human body," Kiev, 1900) of Meyer and Quain (Quain's Anatomy, 1866; London).

Tichomiroff's scheme of anastomoses of the ophthalmic art. is as follows:—

(a) From the trunk of the art. carot. interna are branching off twigs, going to the fissura orbital: ophthal. superior and to the trunk of the n. opticus; here also are coming the twigs from the trunk of the ophthal. artery, and they anastomose one with the other, and so form a link between the two arteries. A good illustration is the variant described by Meyer (illustration 4, Fig. 7b, r.a.c.).

(b) The ophthalmic artery supplies twigs to the trunk of the n. opticus, that anastomose with one another, the proximal ones going in the distal direction and *vice versa*. An illustration of this is the same figure 4 (7b, r.a.o.).

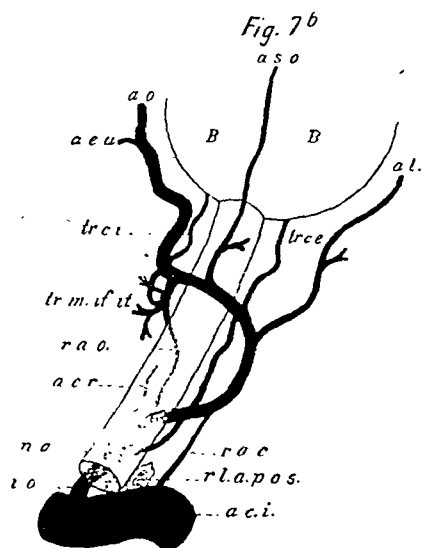


ILLUSTRATION 4.

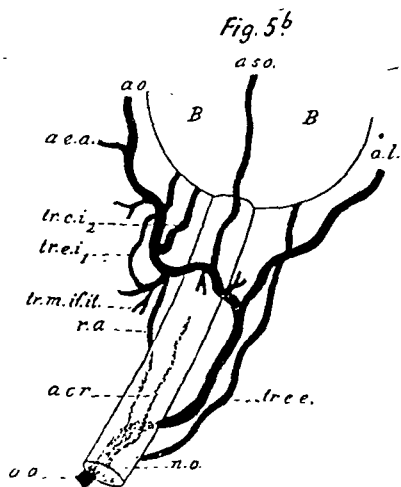


ILLUSTRATION 5.

Illus. 4, Fig. 7b (reconstituted normal route)—r. a. c. an anastomotic branch between ar. cen. int. and art. ophthalmica, passing through fiss. orbit. superior, r. a. o.—an anastomotic twig between the branches of the ophthal. art.—it passes under the n. opticus; a. c. i.—art. carot. interna; a. o.—art. ophthal; tr. c. e. extern. branch of the art. cil. post. long. tr. c. i.—the internal branch of the same artery; a. c. r.—central retinal artery, a. l.—lacrymal artery; a. s. o.—supra-orbit. artery.

Illus. 5, Fig. 5b. (reconstituted normal route)—r. a. anastomotic branch between the branches of the ophthal. art. and it passes under the n. opticus; a. o.—ophthal art.; tr. c. e.—an external twig of the art. cil. post long. tr. c. i.—the internal twig of the same art., a. c. r.—central retin. artery, a. l lacrym. art; a. s. o.—supra-orbit. art. F. Meyer, "Zur Anat. Der Orbitalarterien."

(c) Twigs from the central ret. art. going to the trunk of the n. optic. form an anastomosis with the similar ones from the ar. ciliar. longa posterior. This variant is also described by Meyer (see illustration 5, Fig. 5b, r.a.).

(d) Anastomosis with the end of the art. maxillans externa (a. angularis) through the nasal branch (ram. nasalis) of the ar. naso. frontalis (ram' art. ophthalmica).

The same variation is represented in the illustrations of Quain, Testut, Vorobjeff; one of them borrowed from Quain is represented here; illustration 6 (IV, 10).



ILLUSTRATION 6.

Illus. 6 (half-schematic ?) I—ar. carot. interna; II—art. basil.; III—the upper end of ar. carot. ext., III', III"—arteria maxill. interna; its branches:—1—art. alveol. inferior; 2—ar. mening. med.; 3, 3—art. masseter. et pterygoidea 4.—ar. buccinat; 5, 5—art. temp. profund. 6—a. alveol. sup.; 7—a. infraorb.; 8—the final branch of art. max. inferior; 9—art. max. ext.; 10—a. angul.; 12—a. nasal. later. 11, 11'. Vessels of the orbit 1, a. ophthalmica; 2—a. lacrimalis; 3—a. centralis retinae; 4, 4—a.a. ciliares posteriores, 5—a. musculares, 6—a. supra-orbitalis; 7, 7—a.a. ethmoidales posterior et anterior; 8, 8', 8"—a. palpebralis; 9—a. frontalis; 10—a. nasal a., which forms an anastomosis with the angular artery (Quain).

(e) An anastomosis of the art. tempor. profund. ant. with the lacrymal and muscular branches of the ophthal. artery; this anastomosis is made through the orbital twigs of the temporal artery; they go through the fissura orbital inf.: and through the temporal canaliculus (see illustration 6, IIL, 5; 5), Quain.

(f) Entering the orbit through the fissura orbital super. the branches of the arter. mening. med. form an anastomosis with the twigs of the lacrymal artery. This variation is demonstrated by the illustrations of Meyer and Adachi (see illustration 7, Fig. 3b, r.a.m., Meyer, and illustrations 2, Fig. 63, Adachi).

Here it is worth while to speak about 2 other variations that occur with the participation of the ar. mening. media—we spoke

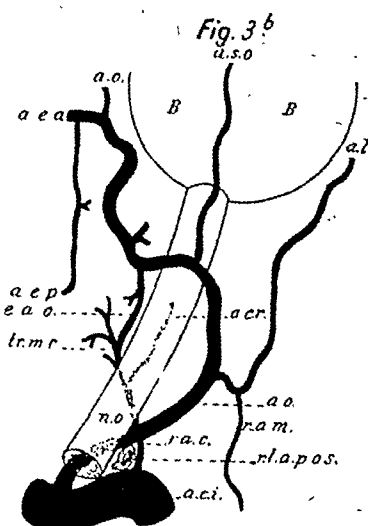


ILLUSTRATION 7.

Illus. 7, (Fig. 3b) (reconstituted normal route) r.a.m. anastomotic branch between the lacrymal art. and art. mening. media, that passes through the fissura orbit. superior, r.a.o.—anastomotic branch between branches of the ophthalmic artery, r.a.c.—anastomotic branch between the a. carot. int. and ophthalmic artery, that passes through fissure orbitalis superior; a.c.i. carotis int.; a.o.—a. ophthalmica; a.l.—a. lacrimalis; a.s.o.—a. supraorbitalis; a.c.a., a.c.p.—aa. ethmoidales anterior et posterior.—F. Meyer.

about them in the beginning of this paper (see illustrations 1 and 3).

(g) The orbital branches of the ar. infraorbitalis form an anastomosis with the muscular branches of the ophthalmic artery (see illustration 6, III, 7, Quain). F. Meyer, when describing the various positions of the ophthalmic artery, points out the cases where the artery crosses not above, as usual, but below the optic nerve (also Zuckerkandl, Merkel, Adachi, Taguchi, Javle and others). Merkel's "Macroscopische Anatomie," Graefe-Saemisch, Aufl. I, Bd. 1, T.I. (Cap. 1, 1874), describes vaguely the similarity between the arch of the aorta and some cases of the ophthalmic artery, when its medial part unexpectedly finishes in the posterior ethmoidal artery and so terminates behind the eye instead of going forward, as usual.

When discussing the cause of the above described various positions of the ophthalmic artery, Meyer and others found it in the atrophy or under-development of the main trunk of the opht. art. Meyer even constructed a schematic location of such an atrophy, guided chiefly by variation of the lacrymal and supra-

orbital arteries; he differentiates three positions of the possible localisation of the atrophic main trunk of the ophthalmic artery—*first*, up to the lacry. art. (Fig. 5a—X; Meyer, illustration 8); *secondly*, between the lacrymal and supra-orbital artery (Fig. 7a,

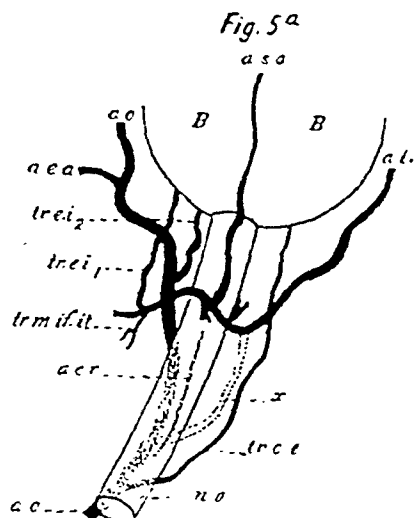


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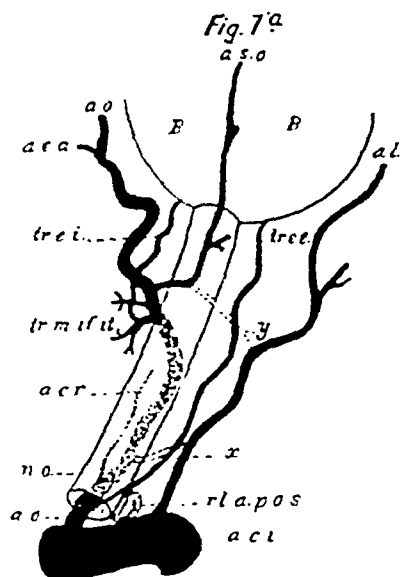


ILLUSTRATION 9.

Illus. 8 (Fig. 5a) Natural state; x—the part of the trunk of the opth. art. that disappeared; a.o.—a. ophthalmica; tr. c.e.—the external branch of the a. cil. post. long; tr. c.i.—the internal branch of the same art. a.c.r.—a. centralis retinae; a.l.—a. lacrymalis; a.s.o.—a. supraorbitalis.

Illus. 9 (Fig. 7a) Natural state; x and y—the parts of the trunk of the opht. art. that disappeared a.c.i.—a. carotis interna; a.o.—a. ophthalmica; tr. c.e.—the external branch of ar. cil. post long; tr. c.i.—the internal branch of the same art. a. cil. p.l.; a.c.r.—a. centralis retinae; a.l.—a. lacrymalis; a.s.o.—a. supraorbitalis.

Y; Meyer, illustration 9); *thirdly*, more distal from art. sup. orbit. (Fig. 4a, X; Meyer, illustration 10).

He reckons, that at the same time—and in the same case—two such atrophic parts may appear, one before and the other one after lacrym. art. (Fig. 7a, X-Y; illustration 9), and even all three together. (He never saw it himself, but thinks it is possible.)

But Meyer did not explain what is the cause of such an atrophy of the trunk of the opththal. artery, nor in which period of life does it start; we can only suppose that it happens in the embryonic period, or in the very early post-embryonic period (a very young patient, a female, Meyer).

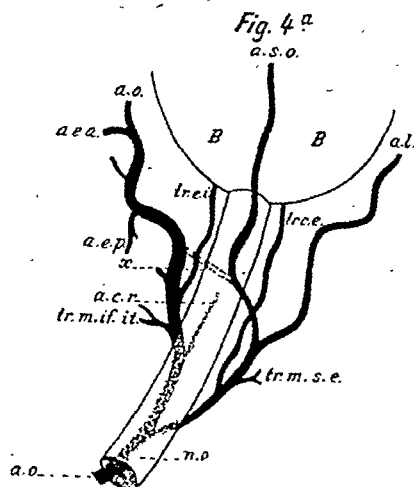


ILLUSTRATION 10.

Illus. 10 (Fig. 4a) Natural state; x—the disappeared part of the trunk of the ophthal. art.; a.o.—a. ophthalmica; tr. c.e.—external branch of the ar. cil. post long; tr. c.i.—internal branch of the same art; a.c.r.—a. centralis retinae; a.l.—a. lacrimalis; a.s.o.—a. supraorbitalis; tr. m.s.e.—the superior-external muscular branch; tr. m. if. it.—the inferior internal muscular branch.

As far as the connection of the different variations of the branching of the ophthal. artery with this atrophy, Meyer remains the partisan of the old theory and describes the variations of the lacrymal and sup. orbital arteries. The ciliary arteries and even the central retinal artery are left here on the second plane, and are brought forward only when discussing the question of the first branch of the ophthalmic artery.

And so all the researches of Meyer—very valuable indeed—show some detachment of anatomy from physiology and without answering the question (I will speak about that further on), make all the anatomical progress in this direction fruitless. So it happened with Meyer—so is the position to-day.

After all the previous remarks, we will give a short description of our own anatomical studies, with special attention to the two most essential points—the system of the ophthal. artery in the orbit itself and its part within the skull. The ophthalmic artery forms an angle when it goes round the optic nerve before it enters the foramen opticum; first below it and externally, then to the superior external margin and then it turns towards the medial side and crosses the optic nerve from above.

This angle exists nearly always. As a rarity it may be absent, for instance, if the ophthalmic artery is situated below and to the medial side of the optic nerve. The angle is not always the same, not only in different persons, but even in the same person. In childhood and adolescence the angle is always a sharp one. After 24 years of age, it is chiefly perpendicular, and after 40 years it is an obtuse one. The sharp angle may be preserved, in single

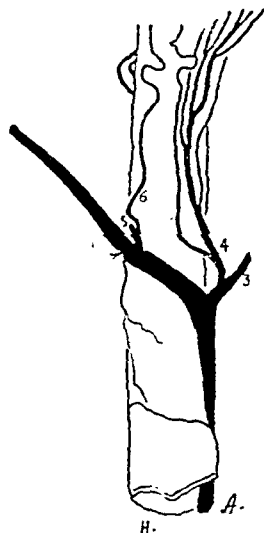


ILLUSTRATION 11.

Illus. 11 (magnified) right eye view from above optic nerve. A—ophthalmic artery; 3—a lacrymal artery, branching off on the site of formation of the first angle of the ophthalmic artery, 4—lateral branch of the art. cil. post. long; 5—the superior external muscular branch and a twig; 6—ar. cil. p. brevis (Male, 4½ years old).

instances, up to senility, but we do not see it any more after 65 years of age.

We think that nobody up till now has described this angle, but the variations of this angle that are due to age change the correlation between different parts of the ophthalmic artery and so the direction of the pulse-wave, because the artery in this particular spot is always firmly fixed to the membranes of the optic nerve and cannot straighten its angle every time in connection with the pulse-wave. It is because of this that the angle changes with age from a sharp to an obtuse one.

This is the result of two constant but opposed factors—one force working in the distal, the other in the proximal direction. If we imagine that these two forces are acting along the axis of the artery, then the vessel will gradually become longer, forming

bends, and these bends will be evident at every arrival of the pulse-wave; Hyrtle already pointed it out—J. Hyrtle, "Handbuch der Anat. der Menschen," 1879. (This locomotive pulse is a physiological phenomenon of the central ret. artery, described by the Translator and others—Translator's remarks.) As an instance, we can point to our fixed preparations and illustrations of the trunks of the posterior long ciliary arteries (illustration 13 and others); we were able to fix the bends of these arteries by injecting them with ether-cellaodine (red praecip. mercury). But if we find as a primary phenomenon an angle of the ophthalmic artery and, moreover, if it is situated in a most fixed part of the vessel, then we can explain it only by a lateral action of a force usually directed from forward backwards. This force may come only from the branches of the ophthal. artery, situated distal to the described angle. These could hardly be the muscular twigs, because they are usually of equal force and are directed in mutually opposed direction. Therefore they could be only the sup. orb. art. and the posterior ciliary arteries, and probably chiefly the medial branch of the latter. To clear these points, I will cite instances. But before that we have to notice the following: the posterior long ciliary arteries and the central retinal artery usually arise together in the first part of the ophthalmic artery. The first to arise is the medial branch, forming a common trunk with central retinal artery; the

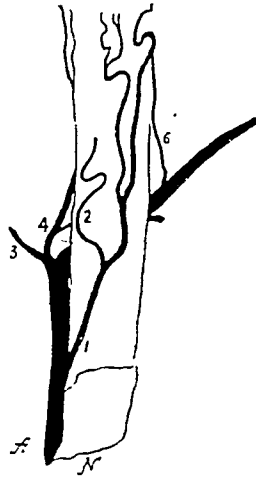


ILLUSTRATION 12.

Illus 12. The same right eye as in the illus. 11. View from below; 1 medial branch of the art. cil post. long; forming a common trunk with (2) central ret. art. 3—a; art. lacrymal; 4—(lateral branch of the ar. cil post. longa.).

second is the lateral branch and in the angle itself is the third branch, the lacrymal artery (see illustration 12). Consequently the long post. ciliary arteries and the central retinal artery receive their blood supply by the shortest way, nearly a direct line. It is really rare to see, in variation, a different way of branching, and in these cases the medial branch is located in the second part of the



ILLUSTRATION 13.

Illus. 13 (magnified) right eye. View from above A—ophthalm. art.; its proximal part is located in the bony canal that is preserved here, forming an obtuse angle. All the ciliary arteries (except the central ret. artery which branches off in the lateral (first) part of the ophthalmic artery—it is covered from above by the optic nerve)—branch off in the second part of the ophthalm. art. (distal to the first angle). 3—lacrym. art.; 4—lateral branch of the art. cil. post. long; 5—muscular branch with branching off it; 6 a. cil. post. brevis; 7—a muscular branch, arising from it; 8 ar. cil. p. brevis; 9—the point of division of the trunk of the ophthalm. art., which forms two lateral branches, going to the medial wall of the orbit—the posterior one going by the usual route of the art. ethmoid. posterior; 10—medial branch of the ar. cil. post. lon. (Male, 65 years of age).

ophthal. artery, and sometimes it is followed there by the central retinal artery. In these cases we can watch the lateral action of the force, as shown by an earlier and more pronounced increase of the angle, gradually changing from a sharp to a perpendicular one, and from a perpendicular into an obtuse one (see illustration 13). Here the medial branch and the rest of the ciliary artery do not receive their blood supply by the shortest way—nearly a direct

line. The blood-wave first goes sharply round, sometimes even with a reverse direction of the flow of blood, and only after that it reaches the medial branch. As a result, the correlation of forces is upset, the trunk of the ophthal. art. is inclined more and more towards the side of the weaker force, and by so doing increases the angle of deflection of the pulse-wave. And so is built the picture illustrated by us. The picture of the obtuse-angle bend of the ophthalmic artery in the extreme degree, as seen in illustration 13 was not seen by us in ordinary circumstances, even in cases of advanced senility (see illustration 12).

This underlines how important is a definite order of branching of the post. long. cil. arteries for sustaining a favourable correlation in the system of the ophthal. artery from the haemodynamic point of view.

In cases of interruption in the trunk of the ophthal. art. (X) before the point of branching of the lacrym. art. (a. 1)—and that can be seen even in illustrations of Meyer (illustrations 7-9), the medial branch of the long. post. cil. art. (tr. c.i.) is not seen between the first branches—in one case it is not shown (Fig. 4a) and in two other cases (Figs. 5a and 7a) it is shown arising from the medial part of the ophthal. art.

Our researches in 14 cases out of 103 did show that in those 14 cases the ophthal. art. crossed below the opt. nerve; in embryonic or early post-embryonic stage there ought to be a defect up to the first angle of the ophthal. artery, but, as a rule, we saw a variation in the usual order of branching, and the medial branch of the long post. cil. art. was found in the second part, after the lacrymal artery (see illustration 24). Therefore we are entitled to two conclusions.

The first being that we can judge about the interruptions (defects) of the ophthal. artery not only according to variations in the origin of the main trunk of the lacrymal and sup. orb. arteries (pointed out already by Meyer), but also on the basis of variations in the rise of the long post. cil. arteries and of the central retinal artery (see our cases, illustration 24), if those defects happened in the part between the foramen opticum and first angle (*i.e.*, before the branching of the lacrym. artery), *i.e.*, where those arteries are usually branching off. The second conclusion is that the unusual rise of the long. post. cil. art. and of the central ret. artery with their appearance on the 2nd part, distal to the first angle, is an unfavourable factor from the haemodynamic point of view. We can easily prove the second conclusion, as nature itself supplies the facts, by providing in those cases anastomoses and accessory branches.

Some time ago Quain affirmed that the posterior ciliary arteries

do not form anastomoses. If he is right—then it is only in ordinary circumstances. But with variations—it is obvious already from the schemes of Meyer and Tichomiroff—this order is interrupted by nature herself—the posterior ciliary arteries do take part in the anastomoses of the ophthalm. artery and form anastomoses themselves as well. Because of these anastomoses a circuitous canal is



ILLUSTRATION 14.

Illus. 14. Magnified, left eye. View from below. A—ophthal. art. with its proximal part concealed in the bony tunnel preserved here; it forms an obtuse angle, similar to Illus. 13. All the ciliary arteries and the central retinal artery arise from the second part of the ophthalm. artery (—distal to the first angle). 1. A powerful lateral branch that supplies 2 ar. cent. retin. and (3—5) 3 post. cil. arteries. 7—a lateral branch of ar. cil. post. long; 6 and 9—lateral branches, the last one supplying (10) a. post. long. cil. arteries; 11—powerful anastomosis between the long. post. ciliar. arteries (between 4 and 10), forming a vascular ring round the optic nerve. The ophthalm. art. does not go forward, as usual, but follows the route of the ar. ethm. post. (Male 65 years.)

formed, joining the first and second parts, bypassing the angle; and the blood-wave through this canal finds a new collateral way towards the posterior ciliary and neighbouring arteries. This is the same closed vascular ring round the optic nerve that was described by B. Sokoloff (B. Sokoloff, "Ciliary arteries in man," Perm., 1939), as the starting point of the ciliary arteries and was named by him "*circulus arteriosus ciliaris periopticalis*." The beginning of such a ring we saw already in the first year of life,

and in this case, the medial branch of the long post. ciliary arteries was situated in the second part of the ophthalmic artery (see illustration 15, 10—A.H.; 8)—medial branch of the long post. cil. arter.

The second factor that we see with such variations—also a compensatory one—is a considerable increase in the number of the short post. cil. arteries; here we see not the usual two, but 3-4 and even 5 branches—and they are located not only in the second, but also in the third part.

Another fact is worth mentioning. The resistance against the blood-wave that we have already described as a force acting



ILLUSTRATION 15.

Illus. 15. Left eye. View from below. A—ophthal. art. whose anterior part. (medial branch of the second part) is divided into two (11) non-equal branches; the posterior—the more powerful one—the real prolongation of the trunk of the oph. art. follows the route of the a. ethmoid. post.; 1—lateral branch of muscular type supplies a. (2) cent. retinae, and (3 and 4) short ciliar-arteries; 5 lateral branch of the long. post. cil. art.; 6—lateral branch of the type of lacrymal artery; 8—medial branch of the long post. cil. art.; 9—lateral branch of muscular type forms an anastomosis (X) between its branches and another anastomosis (10) joining it with a lateral branch of the first part (1). In such a way a vascular ring is formed, that will supply a collateral route for the first and second part of the ophthalmic artery by developing the anastomoses. (A boy of 4½ months).

proximally and at an angle on the trunk of the ophthalmic artery, is helped by another force, acting proximally along its route. This happens in cases of the so-called diffused form—of the ophthalmic artery, *i.e.*, when the medial part of its second part looks like being interrupted and forms 2-3 lateral branches that leave the trunk under an angle (illustrations 19 and 20). The force of resistance, starting here (x) works in the reverse direction vis-à-vis to the blood stream, and shows itself in the initial part of the ophthalmic artery, inside the skull. We are now going to analyse it. (x) By its location this part seems to correspond to the third distal cut in the ophthalmic art. that was described by Meyer (Fig. 4a, X, illustration 10). We think that with this location of the cut in the ophthalmic art. is connected the change of its course from anterior to medial-posterior, that was mentioned by Merkel; we saw it in 5 out of 103 cases (illustrations 19 and 20).

Here in the cavity of the skull, in the narrow space between the entrance of the optic canal and medial to processus clinoides anterior are meeting together—the internal carotid artery, the first part of the ophthalmic art., the optic nerve and the edges of the intra-cranial opening of the optic canal. Their correlation may be varied not only in different personalities, but in the same person at differing ages. I allow myself to visualise this meeting point as a triangular figure with equal sides and the base to the front. Its upper part belongs to the optic nerve, its lower one to the upper part of the bend of the internal carotid, the base—to a surface tangential to the bony edge of the intra-cranial opening of the optic

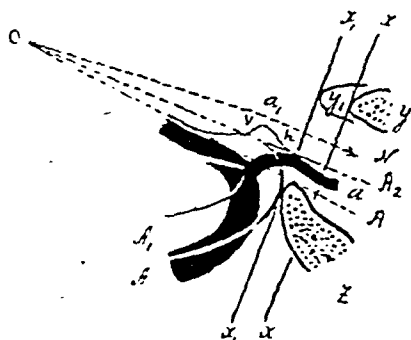


ILLUSTRATION 16.

Illus. 16. A, a—silhouette of the inter. carot. art. and ophthalm. artery in a child; A, a—the same in grown-up persons; y, y—the upper bony edge of the intra-cranial opening of the optic canal; z—its lower bony edge; x, x—lines of the surfaces tangential to the upper and lower edges of the opening of the canal—the surface tangential to the upper bony edge is the base of the triangle; ON—the upper side, OA—the lower side of the triangular in childhood, ON and OA₂ in adults; V—the short knee and X—the long knee of the first part of the ophthalm. art. in adults.

canal. The ophthalmic artery is located inside of this triangle, between its sides and nearer to the base (see illustration 16).

In the beginning the base of this triangle is relatively large. The point of branching off for the ophthalm. art. from the internal carotid and the anterior edge of the arc of carotid, are nearly tangential to the same vertical surface. The angle of the ophthalm. art. branching, that is always larger than 90 deg., looks like a crossing of two arcs—ophthal. and inner carot. arteries. This picture we saw in a mature embryo and in children up to 4 years of age. (Illustration 16, A. a.)

In adults the picture is different. The base of the triangle with age becomes smaller. The lower side (OA₂) gradually disappears,

i.e., the arc of the inter. carotid is gradually and actively straightening upwards and partly forwards. As a result, the sides of the triangle come nearer, and the space occupied by the first part of the ophthalmic artery is limited. Hence follow all variations in the correlation between the blood-vessels, optic nerve and the supporting tissue. The arc of the internal carotid artery (A) takes the form of an angle. The first part of the ophthal. artery (a) recedes 2-3 mm. backwards from the vertical line tangential to the anterior edge of the arc of the intern. carot. art.

The first part of the ophthal. art. is seen already not as an arc, but as broken-line like \square the shorter knee \surd the real start of the ophthal. art.—2 mm. long—nearly preserves the previous relation to the arc of the carot. interna, but this part is perpendicular now to the optic nerve, because the arc itself changed its place; the longer knee—h—passes tangential to the lower edge of the optic nerve.

If we seize the optic nerve with forceps, we can displace it to one side and so approach the initial part of the ophthal. art., or if we press on the arc of the inter. carot. from below, then the optic nerve will be lifted up—we have no obstruction here.

But such a correlation is possible and exists only with a definite structure of the intra-cranial opening of the optic canal, when the edges of both bony prominences are not tangential to one vertical surface (illustration 17, XX; X, X); just the opposite, the upper one—Y—is moved forwards, the lower one—Z—backwards, so that the arc of the inter. carotid is tangential by its edge to the surface of the lower prominence (Z, X, X) and does not reach the surface tangent to the upper one (Y, XX). The ophthal. art. branches off the internal carotid 3-6 mm. backwards from the edge of the upper bony prominence—Y. So we saw it in 9 out of every 10 cases. But in every 10th case, we saw a different correlation. The edges of both prominences are tangential to the same vertical surface (Y, Z, X, X), so that the arc of the internal carotid is tangential by its anterior edge to the lower prominence and to the surface tangent to the upper prominence. The ophthal. art. branches off the carot. int. under the upper bony prominence Y, and the latter forms for it a bony roof.

If in such a case we try to displace with forceps the optic nerve to reach the initial part of the ophthal. art., we will not succeed—the bone that hangs over the artery will be in the way, and also if we press from below on the arc of the internal carotid art.—our pressure will be transmitted to the optic nerve, but the latter will not rise over the artery—the same bone will not allow it—as it will be only pressed against the bone. This initial part of the ophthal. art. in such case will be squeezed in the narrow space

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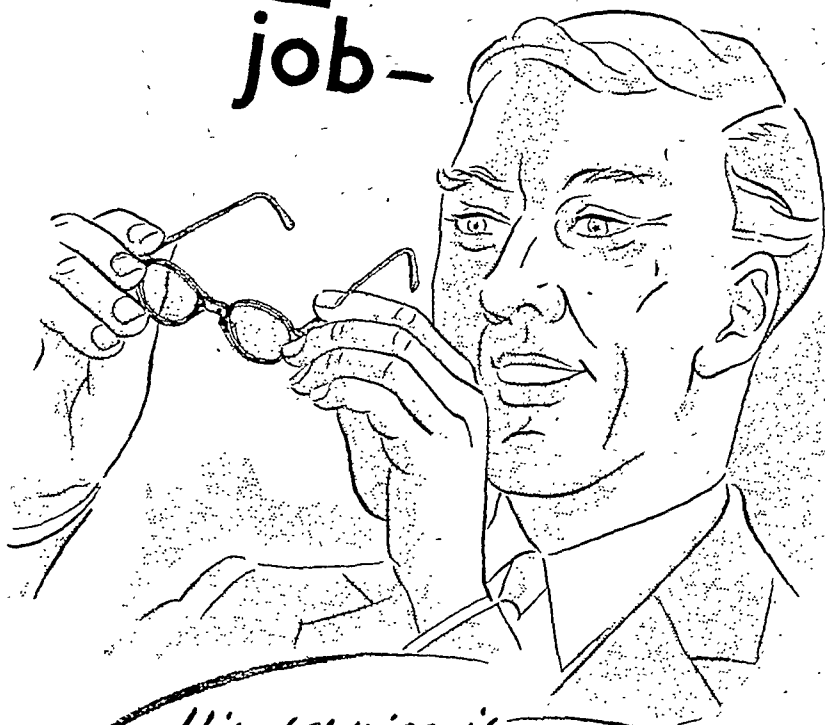
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between the sides of the triangle; i.e., between the opt. nerve and the upper knee of the arc of the internal carotid.

And, in our opinion, such a state is important, not only for the function of the ophthal. art., but even for its existence. This shows itself by the changes in the external architecture of the internal carotid and ophthalmic arteries, and by the changes in their lumen.

Let us study it more closely. Ordinarily the difference in the circumference of the carot. ar. before and after the branching off of the ophthal. art. is 2-3 mm., and does not exceed 4 mm., but in our cases, in 5 out of 10, the difference is 5 and even 8 mm. The increase in this difference is due to the increase in the size of the internal carotid before the branching off of the ophthal. art. Because of that the carot. int. may look like an ampoule (in one of our cases).

The ophth. art. on the other hand was smaller inside and was only 4-4.3 mm. Externally the art. had additional bends in the first part—it looked lifeless; and the branches—the posterior ciliary arteries—were abundant in angular bends instead of forming arcs (illustrations 17 and 18).

Finally the lumen of the carot. int. showed here an earlier and more intensive agglomeration of sclerotic spots that formed massive layers and sometimes even a whole tube of bony hardness with a tiny opening for the branching off of the ophthal. art. The lumen of the ophthal. art. is narrow, the usual needle of a 2 gramme syringe could not enter it freely, as usual. And sometimes it went so far, that the initial short knee of the ophthal. art. had only the lumen of a capillary. From 10 of our cases, where the ophthal. ar. branched off the carot. int. under this bony roof, in two we found this capillary lumen of the initial part of the ophthal. art. (illustrations 17 and 18).

We can look upon this capillary lumen of the ophthal. art. as an incomplete intracranial cut. Such an instance has already been described by Meyer for the intra-orbital part. But in our opinion, his cases and the cases (especially the consequences) that we saw not only between adults, but even in infants 3-4 months old, are really of embryonic origin.

The same intracranial interruptions of the ophthal. art. that we were the first to see ought to be reckoned according to its genesis, not only post-embryonic but even due to the age, because they appear together with senile arteriosclerosis. The immediate cause of their appearance is obvious from the above described architectural changes under a bony roof. The mechanism of these variations is as follows: The initial short knee of the ophthal. art. —V—as described above, is perpendicular to the optic nerve. Because the systolic-wave, knocking at the upper wall of the artery,

tries to straighten the artery out, but meets the immense obstacle of the bony roof, it loses part of its locomotive energy before it penetrates further into the ophthal. artery. But this lost energy does not disappear without trace—it is transformed qualitatively into a new force—into a pressure, that is transferred back into the arc of the internal carotid, and so increases the intra-arterial pressure of this artery, and this is fairly high in any case. So are created favourable conditions for the local changes, that we have just described.

The capillary lumen, or in other words, an incomplete interruption of the initial part of the ophthal. art., due to age, starves the

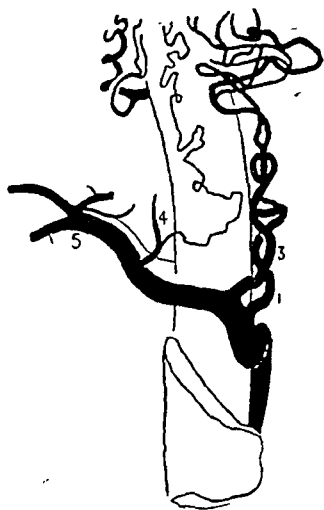


ILLUSTRATION 17.



ILLUSTRATION 18.

Illus. 17. Right eye. View from above. The ophthal. art. in its first part has an additional bend; the medial part of the second part (5) is divided into 2 unequal branches, directed towards the medial wall of the orbit, and the more powerful posterior one is really a prolongation of the trunk of the ophthal. art. and follows the route of the post. ethmoid. art. 1—a medial branch of the art. cil. post. long. arises, as a first branch by a common trunk with the central retinal art. (covered from above by the optic nerve). 3—lateral branch of the ar. cil. post. long. 4—a lateral branch with a small twig towards the eye.

Illus. 18. Left eye. View from above. The ophthal. art. with an additional bend in its first part, rather weak in its size and appearance; its medial part, in the second part, in spite of its bend, still preserves the magistral type and its forward direction. The order of branching is similar to illus. 17—the first to arise is the medial branch of the art. cil. post. long. by a common trunk with the central ret. artery (covered from above by the optic nerve in such a way that only the dorsal part of the medial twig is seen (1); 3—lateral branch of the ar. cil. post. long. In both cases the weakness of the branches, multiplicity of their bends formed with an angle instead of the usual arc, and the increase in their length are remarkable (female, 80 years of age),

whole system of its chief source of blood supply, and it ought to cause it to perish. But this is an experiment *sui generis* staged by nature herself, probably not known to Wagenmann when in his experiments he cut across all the posterior ciliary arteries in a rabbit and sometimes produced phthisis bulbi (Martin Bartels—"Blutgefasse des Auges bei Glaukom"—Diss, Berlin, 1905). And this experiment of nature looks even more dangerous than the cutting of the ciliary arteries. But we never saw in such cases a phthisis bulbi, in spite of the fact that we are certain that this capillary lumen existed much earlier than our discovery. How then otherwise can one explain it, if not by collateral blood-supply, formed out of permanent anastomoses of the ophthal. art.? And all depends only on the ability of those anastomoses to replace the interrupted route of the blood-supply.

We judge the sufficiency of the development of the ophthal. art. anastomoses as we did before by the development of the variations of the basic magistral; it would correspond to the law of Krause, mentioned already by Meyer. The law of Krause says, that variations arise because of wrong development of normal anastomoses; and according to this law one would suppose that the anastomoses of the ophthal. art. would be developed better in the case 64 (illustration 17), than in 64a (illustration 18). And the external view of the arteries in both cases follow this law—they look better in the first case than in the second one, where we see relatively less variations in the basic magistral.

And so on the basis of our cases we are convinced of how important in the life of every human being is the definite osseovascular correlation, which develops in the narrow intra-cranial space in front of the optic canal.

We established the important part of the bony roof as a direct cause of an interruption of the ophthal. artery at certain ages.

But this is not the only cause of such cuts. We saw such an interruption with the normal osseovascular relation in this spot in the case of the so-called diffused form of the ophthal. art. described previously; especially when the medial branch of the second part of the ophthal. artery, in the orbit, looks as if being cut, but really divides into three branches, going in opposite directions to each other, and all of them perpendicular to the main trunk. See illustrations 19 and 20 in the case 68a; it appears that such an architecture creates conditions analogous to those, that (or the sequelae of those) we saw in the initial intracranial part of the ophthal. artery, under the bony roof of the intracranial opening of the optic canal. The mechanism of the action, different in form, is essentially the same. Here, in the case of A. 68a, we met an obstacle of bony consistency that narrowed the lumen of

the initial short knee of the ophthal. art.—V—in its whole length of 2 mm.

There was only a capillary lumen in the centre of this blockage, hardly visible to the naked eye. This so-called “due to age”—incomplete interruption of the ophthal. art. ought to cause the destruction of the whole system. But we did not see it happening.

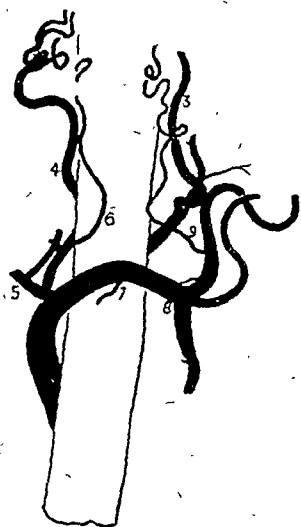


ILLUSTRATION 19.



ILLUSTRATION 20.

Illus. 19 and 20. Left eye. View from above and below. The ophthal. art. goes round the optic nerve by a powerful arc, and medial to the nerve in the second part, suddenly comes to an end (8) and forms 3 lateral branches, that go in different directions with a perpendicular angle. 1—powerful muscular branch, from which branch off (2) cent. ret. art., and later a medial twig of the ar. cil. post. long. (3); 4—a lateral branch of the a. cil. post. long.; 5—a lateral branch of the type of the lacrymal art., that gives off a lateral branch of 2nd degree with a small twig attached; 6—ar. cil. p. brevis; 7—lateral branch; 8—the point of dividing of the main trunk of the ophthal. art.; 9—a. cil. p. brevis. (Female 45 years of age).

The external architecture of the arteries gives evidence of their good tonus, their good general state. The presence of variations of the ophthal. art., according to Krause's law, points to a good local development of the anastomoses.

If you compare those 3 cases with the “age” interruption of the ophthal. art., then in the latter, we have to acknowledge (68a)—judging by the development of the variations—a better state of anastomoses, than in the other 2, and in those 2 the second one (64a) has the worst anastomoses.

And so, watching the cases of “age” interruptions in the initial

intracranial part of the ophthal. art., we realise the important rôle of the anastomoses of the ophthal. art., and we begin to understand their importance.

Let us return now for a moment to the question of the branching of the ophthal. art. and, following in the footsteps of the previous authors, try to contrast an ordinary scheme of this branching—then as a first branch we have to nominate the medial one of the long post. cil., art., forming a common trunk with the central ret.

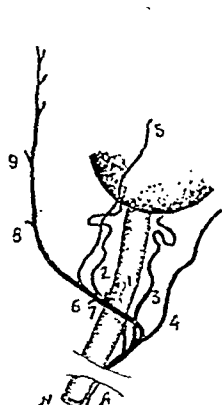


ILLUSTRATION 21.

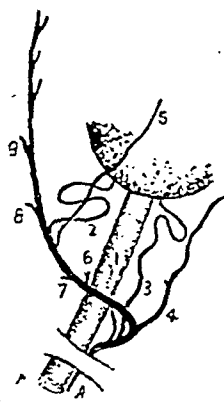


ILLUSTRATION 22.

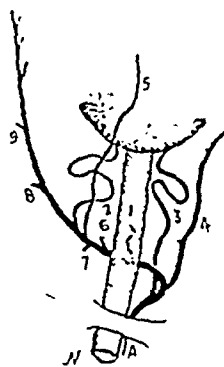


ILLUSTRATION 23.

Illus. 21. Usual type (48·5 per cent.). Art. cil. posteriores and art. centr. retin. branch off together in the first part of the ophthalm. artery. The first to branch off is the medial twig of the art. cil. post. longa by a common trunk with the ar. centr. retin. The central retinal artery has the magistral type with the usual forward direction and crosses the optic nerve from above.

Illus. 22. Variation. The central ret. art. arises first, as the first branch (19·3 per cent.); the long post. cil. art. arise separately, and their medial branch is located in the second part of the ophthalm. artery.

Illus. 23. Variation. The lacrym. art. arises first (13·3 per cent.); all the others—the cent. ret. art., and the long post. cil. ar. branch off it.

art., instead of the central ret. art. according to Meyer, or lacrymal artery according to Testut.

It does not mean, of course, that other branches cannot be the first ones. If we try, guided in each case by the first branch, to divide all our cases in groups, we will have 4 groups. The most numerous one will be the group with the medial one, as the first branch (50 out of 103 or 48·5 per cent.). Then follow 3 weaker groups, nearly equal to one another—2nd with central retinal artery, as the first branch (20 out of 103—19·3 per cent.); third with a lateral branch (19 out of 103—18·4 per cent.) and the fourth one—with the lacrymal artery (14 out of 103 or 13·5 per cent.). For illustration we include a scheme of 7 illustrations (21-27).

In the first 4 we see variations of branching (in one of the illustrations showing the lateral branch, as the first, one can also see the usual in such cases variation in the position of the ophthal. art.—going below, and not above the optic nerve—usually in one eye only in all our cases). The remainder of the 3 illustrations show the variations of the position of the ophthal. art. going across the optic nerve medially and from below, interruption of the medial

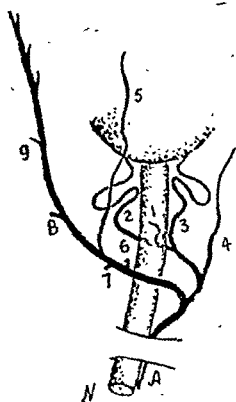


ILLUSTRATION 24.

Illus. 24. Variation. The lateral branch of the long post. cil. art. comes off first (18.4 per cent.); the central ret. art., and the rest of the long post. cil. arteries arise separately and in different parts of the ophthal. art. The ophthal. art. has a magistral type; goes forward as usual, but crosses the optic nerve from below instead of above (13.5 per cent.).

part of the ophthal. art. with an incomplete forward direction—and in the last one, a complete change of direction from a forward one to a posterior medial one and the main trunk of the ophthal. art. going the way of the post. ethmoid art.

A scheme of variations in the location and branching of the ophthal. art., according to our researches is shewn in 103 cases, illustrations 21-27, I-YII.

The basic magistral of the ophthal. art. is marked by the letter A; its branches—by numbers, the same as in all illustrations, 1—a cent. ret. art., 2—medial branch of the ar. cil. post. long; 3—lateral branch of the a. cil. post. lon.; 4—lacrym. art.; 5—sup. orbit. art.; 6-7—muscul. art.; 8—post. ethm. art.; 9—anter. ethmoid art.; 10—the point of division of the basic magistral. Right eye, view from above.

The last variations make the human ophthal. ar. similar to that of the animals, where we always saw the art. end behind the bulb and dividing into two powerful branches—post. cil. art. (see illus. 28)—the result in 50 cases of pigs' and cows' eyes.

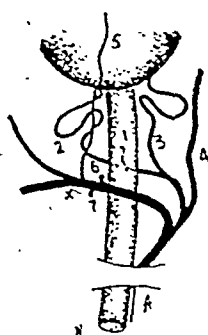


ILLUSTRATION 25.

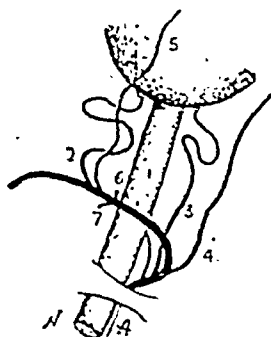


ILLUSTRATION 26.

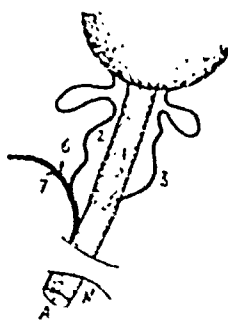


ILLUSTRATION 27.

Illus. 25. Variation. (6.79 per cent.); the ophthal. art. lost the magistral type; the medial part of its second part is divided into 2-3 lateral branches, out of which the weakest goes forward, as would the magistral, and the more powerful one goes directly to the medial wall of the orbit, following the route of the posterior ethmoid. art.

Illus. 26. Variation (4.8 per cent.); the ophthal. art. lost the forward direction, goes to the medial wall of the orbit, following the route of the posterior ethm. art. (may divide itself or not).

Illus. 27. Variation (0.97 per cent.); the ophthal. art. passes under the optic nerve in the same direction, as illus. 26.

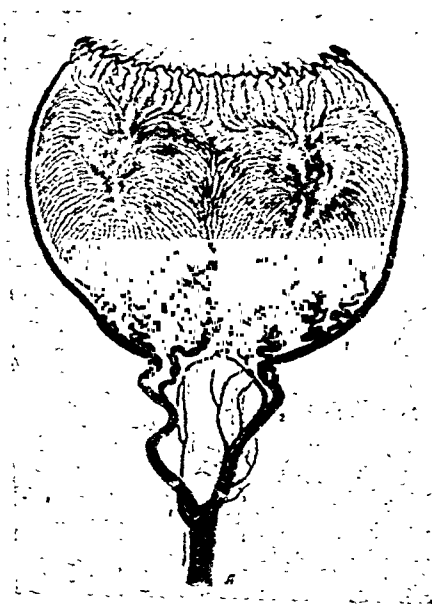


ILLUSTRATION 28.

Illus. 28. Schematic design of the ophthal. art. of the cow and pig. A—ophthal. art.; it gives at first a twig to the optic nerve (1) art. cil. brevis, and later divides in 2 branches—(2) post. long. cil. arteries, and they give twigs to the choroidea—art. cil. post. brevis (3)—in the net of the choroidal vessels two vortices are visible.

And so, coming back to our question, we have to acknowledge that the first branch most frequently is the medial one of the post. long cil. art. with a common trunk with the centr. ret. art. But it will not be the "norm" that was looked for so unsuccessfully by Meyer, it will be only the usual and more frequent order of branching, purposeful as far as all the long post. cil. art. arise together in the first part (up to the first angle), and this gives them a great privilege from the haemo-dynamic point of view. But this purposefulness is only relative and may be changed in the reverse position, when the correlation with other tissues (for inst. the supporting one) will be changed, as we saw in our 2 variations with the "age" interruptions of the ophthal. art. (illustrations 17 and 18, cases 64 and 64a).

Here, in both cases, the first branch was the medial one of the long post. cil. art. by a common trunk with the centr. ret. art.; it looks like a "norm," but both of them, although in a different degree, proved to be unsatisfactory with appearance of the interruption due to age, because both of them probably possessed only weak anastomoses. At the same time, the third case of the same group (illustrations 19 and 20, case 68a) had at first quite an extraordinary one—a muscular artery, that even did not enter into our scheme—and it was quite successful in dealing with such an interruption, because in passing through embryonic variations it was provided with good anastomoses and they were brought into action, when needed.

TRANSPLANTATION OF VITREOUS*†

A Preliminary Report

BY

HERBERT M. KATZIN AND JOHN BLUM

NEW YORK

Introduction

THIS study was stimulated by the recent work that has been done on the transplantation of vitreous particularly by Cutler¹. Many attempts have been made in the past to remove vitreous without replacement or to replace it with saline solution, air, or animal vitreous. With the advent of the Eye Bank, and the availability of fresh human vitreous, we determined to conduct further studies. The

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present report is of a preliminary nature and we expect to amplify the studies with human material at a later date.

Literature

We shall not attempt to make an exhaustive survey of the literature as this will be published elsewhere with the clinical paper that is being prepared on the subject (Fritz²).

In 1890, Ford³, reported on the Proposed Surgical Treatment of Opaque Vitreous. He withdrew from ten to eighteen minims of vitreous through a needle inserted above or below the external rectus muscle. The procedure was repeated in three or four days and vitreous was withdrawn as many as four times. The amount withdrawn depended upon the consistency of the eyeball. Ford assumed that clear vitreous was resecreted.

In 1893, Deutschmann⁴, reported a few good results from the injection of animal vitreous into human eyes for the treatment of retinal detachment.

In 1911, Elschnig⁵, extracted vitreous and replaced it with saline as a method of replacing pathological vitreous.

In 1912, Rosentstein⁶, carried out forty-two vitreous aspirations on twelve patients who had traumatic vitreous opacities, disseminated choroiditis and iridocyclitis, and he observed no ill effect from the procedure.

In 1912, Komoto⁷, "washed out the vitreous" in two cases of eyes that were blind due to vitreous haemorrhage. He made a scleral incision and washed out with a 0.6 per cent. solution of sodium chloride.

In 1921, Erlanger⁸, removed 0.5 to 0.7 c.c. of vitreous and repeated it in ten days on an eye which had an infected Elliot's trephine. The procedure saved the eye with some vision.

In 1921, Blatt⁹, did vitreous aspirations on four patients, removing 0.6 to 0.8 c.c. with satisfactory results. He mentioned as indications, all forms of vitreous haemorrhage due to trauma and uveitis.

In 1922, Blaisch¹⁰, performed three punctures on successive days, removing 0.4 to 0.6 c.c. each time on an eye that had an intra-ocular infection following a perforating injury; the vitreous cleared rapidly and the eye was saved with corrected vision of 6/18.

In 1924, Hamburg¹¹, reported on fourteen cases of chronic iridocyclitis treated by vitreous punctures and aspiration. He reported good results in approximately 1/5 of his cases.

In 1924, Róchat¹², experienced failures in three cases using vitreous extractions for chronic uveitis.

In 1924, Bleidung¹³, reported on sclerotomy without vitreous aspirations in vitreous opacities or bleeding.

In 1925, Zirm¹⁴, did vitreous aspirations on four cases with

vitreous opacities. The result was good in one case, and the other three were doubtful.

In 1927, Bufili¹⁵, presented seven cases of vitreous extraction with good results. They were cases of vitreous haemorrhage, opacities and glaucoma.

In 1928, zur Nedden¹⁶, used aspiration of the vitreous in cases of ectogenous infections, vitreous bleeding, diffuse choroiditis, traumatic and haemorrhagic glaucoma and embolism of the central retinal artery, but he did not replace the aspirated vitreous.

In 1946, Cutler withdrew and replaced vitreous with vitreous from human donor eyes. In two out of three cases in which there was old haemorrhage, the transplant was successful in reducing the opacity.

Methods and Materials

Rabbits were used because of ease of handling and convenience. Thirty-nine eyes were experimented upon and several methods were employed to study the transfer of vitreous.

Group I. In five eyes the vitreous humour was withdrawn without replacement. In these eyes 0.5 to 1.1 c.c. vitreous was withdrawn through a 20 gauge needle at six o'clock, 5 mm. from the limbus.

Group II. In six eyes, the vitreous was withdrawn and saline was injected. In these eyes, 0.7 c.c. vitreous was withdrawn through a 20 gauge needle at six o'clock, 5 mm. from the limbus and replaced with sterile saline by injection through the same needle.

Group III. In four eyes 0.15 to 0.2 c.c. aqueous was withdrawn through a 26 gauge needle which was passed through the limbus and the fluid volume of the eye was restored by the injection of vitreous with a 20 gauge needle through the sclera at six o'clock, 6 mm. from the limbus.

Group IV. Vitreous was withdrawn and replaced in 24 eyes. Three different methods were used:

(a) In two eyes fixation sutures were used to draw the eye over and the region at six o'clock 7 mm. from the limbus exposed. A suture was placed in the sclera, and with a 17 gauge needle 0.5 c.c. of vitreous was withdrawn and replaced and the suture tied.

(b) In fifteen eyes, 0.7 c.c. to 1.0 c.c. vitreous was withdrawn from a donor eye through a 20 gauge needle and injected into the recipient with a 23 gauge needle through the same hole.

(c) In seven eyes, a mattress suture was placed 7 mm. from the limbus in the sclera at 6 o'clock. A 3 mm. incision was made with a Graefe knife between the arms of the suture and from 0.2 to 0.4 c.c. was withdrawn through a 20 gauge needle and injected simultaneously through a 23 gauge needle. The suture was drawn taut and tied as the needle was removed.

Results

Group I. In these five eyes the tension became very soft immediately after withdrawal of vitreous but returned to normal after two to four days. The vitreous became hazy, subsiding during the first week. In three cases, retinal detachment occurred with a hole noted at the site of injection. In two of these, the detached areas subsided within the first month and healed with a small area of fibrous tissue proliferation, forming a retinitis proliferans. In the third case, the detachment was still present on the fourteenth day and the rabbit died on the seventeenth day. Of the four cases that survived, all eventually showed pigmentation reaction around the site of puncture and clear vitreous with normal tension.

Group II. Of these six eyes, one remained clear and five developed vitreous haze. Three of those that developed haze also developed complete retinal detachment which did not subside. The other two cleared. After six months the three eyes which did not develop retinal detachment had completely recovered except for pigmentary reaction around the site of injection.

Group III. In these four eyes, a white, convoluted, fluffy opacity appeared at the site of injection immediately after introduction of new vitreous. The opacities cleared in the course of one week, in three instances, and in the fourth instance, it took four weeks. All the eyes remained with clear vitreous, normal tension and the typical pigmentary reaction around the scar.

Group IV. (a) In one of these two eyes, a cataract formed after the operation and the details could not be followed. In the other eye, a haemorrhagic reaction occurred in the region of the incision and cloudiness of the vitreous developed which took two weeks to subside. The vitreous remained clear, thereafter, and there was no detachment, although there was pigmentary reaction around the scar.

(b). In these fifteen eyes, vitreous remained clear in two and opacities developed in thirteen. The opacities were marked in ten and mild in three. Of those that were marked, six cleared within a period of two to four weeks; two cleared in a period of two months leaving an area of proliferated fibrous tissue and two did not clear. There were four retinal detachments, of which two healed and two remained detached (the two with persistent dense vitreous haze).

(c). In these seven cases, the vitreous was not as easily controlled and a certain amount was lost during the process of injection. In one case, a traumatic cataract formed which prevented visualization of the interior of the eye. In one case detachment occurred which subsided after seven days. Vitreous opacities occurred in all these cases and there was a small amount of haemorrhage near the puncture

Group	Total eyes	Retinal detach. which subsided	Retinal detach. which persisted	Pig. reac. with proliferation	Pig. reac with- out proliferation	Vit. haze which cleared	Vit. haze which persisted	Cataract
Group I	5	2	1	2	3	5	0	0
Group II	6	0	3	0	3	2	3	0
Group III	4	0	0	0	4	4	0	0
Group IV	24							
(a)	2	0	0	0	1	1	0	1
(b)	15	2	2	5	8	11	2	0
(c)	7	1	0	0	6	6	0	1

site in three. However, all of the eyes healed with clear vitreous and the usual amount of pigmentary reaction around the site of injection.

The follow-up period was six months.

Comments

It will be noted that the transplantation of vitreous is not without its complications. Although retinal detachments in rabbits are prone to heal spontaneously, nevertheless, a significant number occurred that did not subside. Cataracts occurred due to injury by the operator. The lens in the rabbit's eye is quite large and difficult to avoid when a large amount of vitreous is removed. Vitreous haze, we believe, is due to alteration in the physico-chemical properties of the injected vitreous and the uveal reaction around the puncture site was normal wound healing. No case of chronic uveitis was noted.

In the human, the pars planum approach to the vitreous is considerably easier, the lens is smaller, but the human eyes would not be normal eyes when selected for a vitreous transplant.

So far as the structural change in injected vitreous is concerned, there are undoubtedly significant alterations caused by the passage through a narrow needle, but from a clinical point of view, the transplant may still be successful and in our experience, vitreous transplantation is more successful than replacement with saline. The eye of the host may metabolize the injected vitreous and accept it into its own structure.

Summary and Conclusions

Transfer of vitreous was studied experimentally in thirty-nine rabbit's eyes with a follow-up period of six months. Several types of procedure were used which are described in the text and the comparison between these procedures in the experimental animal and in the human have been discussed.

Two traumatic cataracts were produced, six retinal detachments occurred which persisted and a localized fibrous tissue proliferation around the puncture site occurred in seven eyes. These complications, particularly the retinal detachment, are significant and should be borne in mind in the election of vitreous transfer as a clinical procedure.

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AUTOHAEMO-THERAPY IN HORDEOLOISIS*

BY

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JERUSALEM

AUTOHAEMO-THERAPY seems to fall into oblivion. In this age of sulfonamides and penicillin it is regarded by many as a sort of mediaeval witchcraft. It has been tried and recommended in so many conditions, and the disappointment of bona-fide followers has frequently been so poignant, that it has fallen into discredit even in cases where its action is beneficial beyond doubt.

Autohaemo-therapy has been tried in asthma, serum sickness, urticaria; it has been employed in herpetiform dermatitis and

* Received for publication, July 8, 1947.

pemphigus, in malaria and hemiplegia, and in practically all inflammatory eye conditions including, of course, trachoma and sympathetic ophthalmia. We find enthusiastic reports and thorough disappointment, tepid recommendation or no reference at all in many text-books.

This bewildering state of affairs is due to the fact that autohaemo-therapy has been employed without discrimination in a multitude of conditions, with disappointing results in most of them.

There are some conditions, however, in which autohaemo-therapy is of undeniable value. Its beneficial effect on furunculosis is well known, and the same applies to hordeolosis, but it seems that the great number of alternative therapeutic agents, particularly sulfonamide and penicillin lately, have overshadowed its value and that it is but rarely used nowadays. It might be justified, therefore, to recommend once more this simple and effective therapy which has proved beneficial in so many cases.

A crop of styes, distributed over months sometimes, can be one of the most trying condition, both for patient and doctor. The number of remedies recommended for it shows that none of them is really satisfactory in all cases. To mention some: hot fomentations of course, and incision; ointments, paints, vaccines and auto-vaccines; yeast, vitamin A, tin salts, particularly stannoxyl, and lately the sulfonamides and penicillin. Ultra-violet, infra-red and even X-rays have been called to help. Autohaemo-therapy and autoserotherapy also belong to this incomplete list, and so does the vast number of preparations for parenteral protein shock therapy, beginning with milk.

There can be no doubt that all these therapeutic agents are useful. Local treatment combined with general medication is usually efficient, and, after all, even the most refractory hordeolosis yields eventually to a well directed therapy. But frequently it is taking up much time, it may even incapacitate the patient as in protein shock therapy, the preparation of auto-vaccines is a long, expensive procedure, and sulfonamides are potent but toxic drugs.

This is the reason why autohaemo-therapy seems to be highly recommendable. It is easily applied, keeps the patient on his job and is easily available. As to the results it is felt that they are as good as any—if not better. I have used autohaemo-therapy on a great number of unselected cases. If a patient tells me that he feels his third or fourth stye coming up he receives invariably an intramuscular injection of 10 c.c. of his own blood—and it is very rarely necessary to repeat this after a week or two. I had occasion to observe many of the patients thus treated over a long period and could satisfy myself that the cure was a definite one.

A few case histories shall illustrate this:

(1) Cpl. W., aged 28 years, was an orderly in my hospital who reported sick in March, 1946, stating that he had suffered from styes for the last 5 months, that he actually was never without at least one or two during this time. He had received treatment on the usual lines by my predecessor which relieved the discomfort at the time and made the styes clear up quickly, but they kept recurring.

On examination he presented a mild chronic squamous blepharitis and one discharging as well as two young styes. He felt run down and listless but was otherwise healthy.

One injection of 10 c.c. of venous blood was made into the muscles of the thigh and the patient was instructed to stop all local treatment except hot fomentations. Two days later he reported back. The styes were going down and he felt subjectively better. A week later he reported again with a small styte on one lid just beginning. A second injection of 10 c.c. was made, the styte did not develop but became resorbed and from then on—he was observed for 6 more months—he had no further trouble. After the first injection he volunteered the information—brought forward hesitatingly and diffidently—that he felt physically better than ever before and that he was sure it was no imagination.

(2) Pt. T., aged 22 years. Reported with recurrent styes which had almost incapacitated him for the past 4 months. He had received routine treatment from the Medical Officer in his unit. On examination he presented one styte in the right upper lid which closed the eye and made it impossible for him to do his work. He received one injection of 10 c.c. of his own blood, and was told to stop local treatment except hot fomentations.

He reported back after one week. The styte had disappeared and he felt well. He was seen again after one month when no further styes had developed, and he wrote, after having been posted away, 5 months later that he had not had any more trouble with his eyes.

This list could be extended but would become monotonous. It may suffice to say that autohaemo-therapy has been used for many years, that all cases responded most satisfactorily, and that only rarely more than one injection had to be given.

It is not the purpose of this communication to speculate about the biological mechanism of this therapeutic procedure. It seems correct to assume that the blood, taken from the vein and injected into the muscle of the patient, has the properties of foreign blood perfectly adapted to the biological equilibrium of the receptor organism. It leads to a desensitization through stimulation of the reticulo-endothelial system. It is conceivable that specific antibodies are formed.

All this can, of course, be provoked by any protein shock therapy.

It is emphasized, however, that auto-haemotherapy seems to be simpler, cheaper, less brutal and all the same equally effective as more elaborate procedures.

ANNOTATION

Medical Imposition

Devoted Austenians will remember among many felicitous sentences in her works, that in Mansfield Park, where it is stated that Lady Bertram was the happiest subject in the world for a little medical imposition. This leads one to offer some remarks on a very threadbare subject, *vis.*, how far it is expedient to tell the whole truth to our patients.?

A great deal depends on the type of patient and what is the matter with the eyes. A nervous sensitive patient of middle age, with a speck or two in her lenses and with full visual acuity, need not be told that she has incipient cataract. Should she mention the possibility of cataract developing we should explain that, while it is technically correct to say that she has some signs pointing that way, it will probably be many years before any serious diminution of vision will happen. One needs to be explicit over glaucoma cases, possible growths, and wounds of the eye. In the last case the sufferer will be well aware that all is not well with his eye; but an early glaucoma case with good vision and field will need careful handling. It is quite essential to get such a case to understand that the eye is in a bad way and it would be most improper to inculcate anything approaching a *laissez faire* attitude. Each surgeon must make up his own mind in each case. No explicit rules can be laid down for his guidance; but in general it may be stated that there are occasions where a little "imposition" may be not only desirable, but also permissible. We recall the case of a middle aged lady with some lenticular opacities and full vision, to whom we did not mention the word cataract. About a year later she called on us again and asked if, at her last visit, we had noted anything amiss with her eyes, as she had in the interval consulted someone else who told her she had cataract, and expressed surprise that no one had told her of it. Her vision was still 6/6 and we said it was our opinion that the word cataract should not be mentioned in cases such as her's where no diminution of vision was noticeable. Some people ask to be told the whole truth, and occasionally others will implore us not to frighten them.

It is also well known that patients do not always tell us the whole truth in their histories, and one sometimes meets with a case, more likely in hospital than in private practice, where the results of an eye injury bear no comparison with the trivial cause narrated by the patient. Such a case was a young man who ascribed a ruptured globe with no perception of light to having fallen on the pavement and knocked his eye on the kerb stone. Some months later we met him in prison; but hasten to add that our own presence there was not due to any summary conviction, but purely to the fact that we were asked to go and help a friend, who was the assistant M.O., to provide half-a-dozen old lags with glasses to enable them to see to do their official tasks.

BOOK NOTICE

Diseases of the Retina. By HERMAN ELWYN. Pp. 587 and xl, 170 illustrations, 19 in colour. London: J. and A. Churchill. 1946. Price, 45/-

It is well recognised that many bodily ailments have characteristic manifestations visible by ophthalmoscopy, and the diagnosis of serious constitutional disease is not uncommonly made first, and frequently quite correctly, by the oculist. The author states in his preface that he has aimed at a complete and systematic presentation of the diseases which affect the retina in order to supply the needs of students and practitioners, presumably both of medicine and ophthalmology; to this extent the book is a compromise with some of the inherent disadvantages which such must entail. From the ophthalmologist's viewpoint, it may be questioned if it is advisable or desirable when dealing with an organ whose parts are so closely integrated as the eye, to devote a book of this length to diseases of the retina alone with little and only passing reference to the oecology of the organ as a whole.

As a compromise, however, it must be regarded as eminently successful. The first part is devoted to a consideration of retinal diseases resulting from disturbances of circulation interpreted widely and including blood diseases, diabetes, and certain other conditions such as disseminated lupus erythematosus. Subsequent parts deal with retinal vascular malformations; degenerative diseases on a hereditary basis; inflammatory diseases of the retina (a section in which one may feel that undue emphasis is laid on tuberculosis); retinal tumours; diseases leading to retinal "detachment"; developmental anomalies including macular colobomata, melanosis retinae,

and congenital retinal folds; and finally, radiational injuries from intense light and from lightning. Contrary to generally accepted teaching, it is suggested that some part of the short wave emission may reach and be responsible for damage to the retina.

Since it describes or mentions the fundus changes which have been observed in a whole host of general medical conditions, this book will be welcome to the ophthalmologist. A valuable, but by no means comprehensive bibliography is appended to the section on each disease discussed: through this it is possible to follow up aspects which one may consider too briefly described, such as genetics in the sections on hereditary defects and the pathology of many conditions.

To the general physician too, this book should be useful, and altogether it may be warmly welcomed as a painstaking and carefully thought out contribution to that ever widening field wherein ophthalmology and general medicine are recognised to overlap.

CORRESPONDENCE

POST-OPERATIVE SECURITY IN CATARACT CASES

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRs,—The paper by Mr. T. G. Wynne Parry on "Post-operative Security in Cataract Cases" (*Brit. Jl. Ophthalm.*, September, 1947) is of unusual interest. Dealing with ignorant unco-operative native patients one is never surprised to see the morning's cataract case sitting outside in the afternoon sun, so any method which offers added security in the closure of the corneal wound will attract attention. But surely the shutting off from view of the periphery of the anterior chamber for four or five days or perhaps longer is a disadvantage? A small hyphaema may be absorbed in that time, a point of significance if this complication is to be treated statistically; also an early hypopyon may be missed and time lost before resorting to subconjunctival penicillin.

Relying on rapid sealing of the conjunctiva to the sclera I have for some time been using a subconjunctival approach (*East Afr. Med. Jl.*, May, 1946). Briefly, an horizontal incision less than the width of the cornea is made in the conjunctiva some 5 mm. above the limbus, and through this the conjunctiva is undermined all round the upper half of the cornea, the separation being no more than will allow the blade of a scissors to pass easily. Worth's

muscle forceps is then placed on the limbus at right angles to the 12 o'clock meridian with one blade superficial and the other deep to the conjunctiva. When locked this forceps gives excellent fixation and a keratome can be passed beneath the blades without the globe rotating, or the anterior chamber can be opened with a B.P. No. 15 knife "ab externo." The rest of the operation is on established lines for the "keratome-scissors" section except that the outer blade of the scissors is passed beneath the conjunctiva. I usually place a mattress suture in the conjunctiva before making the corneal section; when tied it puts a certain amount of tension on the conjunctiva and so helps to keep the edges of the corneal wound in apposition.

I believe that the protective cushioning effect of the intact conjunctiva over the corneal wound prevents painful stimuli which must otherwise occur as the eye turns and the wound edges are rubbed by the lid. Certainly patients seem more comfortable than with the Stallard-Liégard corneal suture. As the section is more "limbic" than "corneal," the ultimate result is an invisible scar.

I am, Yours faithfully,

A. J. BOASE.

MULAGO HOSPITAL,

KAMPALA, UGANDA

October 20, 1947.

NOTES

A SLIP insertion in the August and September *Ophthalmic Literature* issues of the Journal drew attention to the subscription for "Ophthalmic Literature" which starts on January 1, 1948. It is £3 3s. per annum, and, if included in the annual subscription to the *British Journal of Ophthalmology*, the total is £5 5s.

We understand from the manager that though orders have come in regularly since the proposal was announced the response on the whole has not been quite up to expectation. The Journal cannot make a present of "Ophthalmic Literature" to its subscribers as Monograph Supplements have been supplied in the past. The former is a regular quarterly production, the latter appear at sporadic intervals and rarely more often than once a year.

There is always a strong chance that a slip insertion may be put on one side, mislaid or forgotten. In such cases re-iteration sometimes bears fruit. We should not like to think that our subscribers were actuated by sentiments similar to those of the Friend of Humanity in Canning's "Needy Knife-grinder":—"I give thee sixpence! I will see thee damn'd first."

* * *

The Leslie Dana Gold Medal **THE Leslie Dana Gold Medal**, awarded annually for outstanding achievements in the prevention of blindness and the conservation of vision, for the current year has been awarded to Dr. Frederick H. Verhoeff, of Boston, Mass.

* * *

**Royal Society of
Medicine, Section of
Ophthalmology
Officers and Council
for Session 1947-48**

President, Harold Levy; *Immediate Past-President*, P. E. H. Adams; *Vice-Presidents*, Sir Stewart Duke-Elder, K.C.V.O., F. W. Law, Air Commodore P. C. Livingston, C.B.E., A. B. Nutt, J. D. Magor Cardell, Charles

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Dates reserved for meetings of the Section. Meetings are held at 5 p.m. (Cases at 4.30 p.m.). 1947—December 11. 1948—February 12, March 11, April*, June 10 (Annual). N.B.—Meetings may be changed to Fridays at 8.30 p.m. If so, due notice will be given in the Diary Card.

P. M. Moffatt, 115a, Harley Street, W.1, A. Seymour Philips, 104, Harley Street, W.1.—*Honorary Secretaries.*

* * *

**Request from a Swiss
Ophthalmic Nurse-
Secretary**

A YOUNG Swiss lady, 27 years of age, is anxious to visit England in the Spring of 1948 in order to improve her knowledge of English.

*Combined Meeting with O.S.U.K.

NOTES

She would like to find hospitality, on "au pair" conditions with an English eye-specialist for six months.

After leaving school she worked in a private hospital at Fontainebleau at surgical nursing. For the past ten years she has been with Professor P. Knapp, eye-specialist, of Basle, as assistant and surgical nurse.

If any of our readers can help in this matter please write to Madlle. T. Mahrer, c/o. Professor Paul Knapp, Klingenthalgraben, 31, Basle, Switzerland.

* * *

WE have received a letter from Dr. Brecher of Bucharest, which owing to paper restrictions, we are unable to reproduce in full. It deals with "welder's conjunctivitis"—the type produced by exposure to an electric arc—he suggests that the disease is toxo-allergic in origin. He found that the symptoms were relieved by bathing the eyes with a solution of sodium thio-sulphate 2 per cent., combined with "chlorate of calcium" 2 per cent. (we take this to be calcium chloride). Whereas with the usual treatment sufferers from this condition were off work for two or three days, by the above method they were able to return to work without any rest period.

Dr. Brecher suggests that his sodium thio-sulphate and calcium chloride lotion would be of use in other toxo-allergic conditions, such as spring catarrh, filamentary keratitis, etc.

* * *

**International
Organisation Against
Trachoma**

THE International Organization against Trachoma was founded officially by decision of the XIIIth Concilium Ophthalmologicum at its meeting at Amsterdam in 1929. It provides a society at the meetings of which matters of scientific, therapeutic, and international interest connected with trachoma may be discussed.

The last general assembly of delegates and members was held in London on April 21, 1939. The proceedings were reported in *La Revue Internationale du Trachome* for July, 1939, and in the British Medical Journals.

A meeting of the executive committee of the I.O.A.T. was held in Paris on May 17, 1947, at 66, Boulevard Saint-Michel. There were present: Dr. MacCallan (President); Dr. Wibaut (Secretary-General); Professor Nordensen (President of the Concilium

Ophthalmologicum); Dr. Ehlers (Secretary-General of the Concilium Ophthalmologicum); Dr. Bailliant (Président de l'Association Internationale de Prophylaxie de la Cécité); Dr. Lavery (Eire); and Dr. Churchill (Secretary-General Adjoint de l'Association Internationale de Prophylaxie de la Cécité). The accounts were scrutinized and passed as correct, and it was decided to hold the next general assembly of delegates and members and the scientific meeting in London in 1950.

The quarterly journal, *La Revue Internationale du Trachome*, which is published in French and in English, is the official organ of the I.O.A.T. Publication ceased during the War, but it is hoped to recommence shortly. Articles by members of the I.O.A.T. may be sent to Dr. Jean Sedan, 94, Rue Sylvabelle, Marseille, Secrétaire-Général de la Ligue contre le Trachome, Editeurs de *La Revue Internationale du Trachome*.

Membership of the I.O.A.T. is by an annual subscription of twenty-five shillings sterling; this may be paid to the account of the I.O.A.T. at the National Provincial Bank, 23, Wigmore Street, London, W.1. Larger donations from ophthalmological societies are invited for the purpose of meeting the general expenses of the organization.

If circumstances permit, *La Revue Internationale du Trachome* will be posted to members as published.

Further information may be obtained from the President, A. F. MacCallan, Westminster Hospital Medical School, 17, Horseferry Road, London, S.W.1., or from the Secretary-General, F. Wibaut, P.C. Hooftstraat, 145, Amsterdam.

* * * *

Abstract of the Will of Thomas Masters, Spectacle-Maker 7th December: I Charles. Thomas Masters, Spectacle-maker, of St. Clement Danes without Temple barre, being sicke in bodie but of perfect memorie laud and praise be given to God . . . Soule into the hands of Almighty God my maker and of Jesus Christ . . . my bodie to be buried in decent Christian buriall. After payment of debts and funerall . . . I give unto my sonne Francis Masters all my estate whatsoever whether it be redie money, bills, bonds, specialties, leases or any thing els to be paid to him when he shall accomplish the age of 18 and in the meantime I request my loving brother Edward Masters to have a special care over my said child and see him well brought upp, to whome I committ the custodie and keeping of my said child and if it happen . . . to dye before he accomplish the age of 18, then I give to my brother Twigden Masters 40s. to make him a ringe. Item. I give to my brethren

John and Richard 20s. a peece. Residue to brother Edward, my executor. Witnesses. John Horne, Thomas Fox, Mary Masters. Proved 9, March 1625. (P.C.C. 37, Hele).

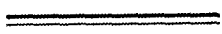
King James the First died in 1625. Masters signed his will on Dec. 7 of that year, and it was proved on March 9th, 1625/26 O.S. It is a pity that he does not mention any of the tools of his craft.

* * * *

Barnacles, Goggles and Gig Lamps THESE terms are all slang for spectacles and like many other slang words are quite descriptive. Barnacles appears to be the eldest of the three. The shorter Oxford Dictionary gives the date 1571 and gives as a probable derivation from the term used for a bit for the mouth of a horse or ass. As the rider bestrides the horse, so the barnacles bestride the nose. Sam Weller made use of the term in alluding to Mr. Ben Allen at Dingley Dell.

Goggle was used in 1688 for a squint, a leer, or a stare. One form of goggles was a form of spectacles, usually with coloured glass, and more rarely of fine wire netting fixed in short tubes to protect the eye from dust; and this form of implement seems to have been used in the treatment of squint. In 1715 spectacles with round glasses were termed goggles; but whether this had any reference to the Old Pretender we do not know.

The use of gig lamps according to the dictionary is comparatively recent, not much earlier than 60 years ago. The idea was derived from the lamps on each side of a gig or dog-cart. Mr. Samuel Pepys, it will be recalled, tried a "tube-spectacall" of paper used with his right eye, and was at first mightily pleased with it. This was in 1668, but the good effects were not lasting. Previously, in 1666, he had bought a pair of green spectacles, but unfortunately for us he does not call them either barnacles or goggles.



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INCORPORATING

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INDEX—NAMES.

vii.

	PAGE
Gördüren, S.	228
Grieve, John	446, 646
Grósz, Stephen de	510
Handousa Bey	155
Haridas, G.	428
Haythorne, Joan	409
Heffernan, E.	427
Hughes, H. L.	689
Irvine, G.	289
James, R. R.	3, 179
Johnstone, I. L.	385
Juler, F. A.	127
Katzin, Herbert M.	760
Kertesy, A.	436, 572
Koch, Walter	235
Krwawicz, T.	421
Landau, J.	145, 219
Lavery, F. S.	125
Lees, P. T.	318
Lees, V. T.	713
Leigh, A. G.	447
Levkoieva, E. Th.	336
Lindsay-Rea, R.	128
Lloyd, Ivor	51
Lyle, T. Keith	191
Lyons, F. Maxwell	703
Macaskill, J.	193
MacCallan, A. F.	711
Magnus, J. A.	692
Mann, Ida	40, 565, 676
Martin-Jones, J. D.	127
Martin, L. C.	129
Michaelson, I. C.	193
Moore, R. Foster	46
Muirhead, C.	319
Nairac, M. L.	700
Neely, J. C.	581
Neubert, F. R.	275, 512, 711
Parry, T. G. Wynne	569
Pascal, J. I.	424, 570
Pearse, R. W. B.	129
Peleska, M.	419
Pollock, W. B. Inglis	123
Preston, F. E.	250

	PAGE
Quilliam, J. P.	717
Raáb, C.	510
Reed, Howard	409, 528
Robson, J. M.	193
Roper-Hall, M. J.	223
Rugg-Gunn, A.	396
Savin, L. H.	449
Somerville-Large, L. B.	192, 362
Sorsby, Arnold	409, 517, 528
Stallard, H. B.	12, 447
Stern, H. J.	373, 766
Stutterheim, N. A.	721
Sudakevitch, T.	738
Taylor, Charles	211
Traquair, H. M.	8, 316, 512
Ungar, J.	517
Vasko, A.	419
Verhoeff, F. H.	774
Walker, J. P. Spencer	383
Weinstein, P.	238
Weve, H.	384
Wilczek, Marian	551
Wolf, Eugene	127, 427
Wood, Philip M.	712
Zondek, B.	145

OBITUARY

Collins, Sir William J., K.C.V.O.	126
Cruise, Sir Richard, G.C.V.O.	125
Lawson, Sir Arnold, K.B.E.	251
McGregor, Ian Stewart	318
Nayar, Dewan Bahadur Koman	253
Thompson, G. W.	711

INDEX—SUBJECTS.

SUBJECTS.

	PAGE		PAGE
Adenoma arising in a sweat gland of the upper eyelid, a case of	697	Appointments	127, 320, 384, 446, 712
Allergy to endogenous hormones as a cause of keratitis rosacea	145	Aqueous humour, pathological changes in the	223
Aluminium and its alloys. The effect on the eye: a report presented to the Vision Committee of the Medical Research Council	449	the influence of intra-ocular pressure on the rate of drainage of. Stabilization of intra-ocular pressure or of aqueous flow	160
Amblyopia, tobacco, and the budget (Annotation)	503	Army recruits, visual defects in	513
American Ophthalmological Society (Officers) meetings, June 1947	504	Arteries, the variations in the system of the trunks of the posterior ciliary	738
Amniotic membrane grafts in caustic burns of the eye, further experience with	409	Astigmatism in Indians (Correspondence)	313
Angeoid streaks of the retina	119	Atebrin workers, "Blue haloes" in (Correspondence 191)	40
Anfridia, a consideration of, with a pedigree	649	Australia, The Ophthalmological Society of	115, 712
Annotations		Autohaemo-therapy in hordeolosis	766
Areopagitical Medical imposition	54	Autonomic nervous system, xanthelasmata and	686
Mister or Doctor (Correspondence 191)	769	Barnacles, goggles and gig lamps (Notes)	776
Moorfields, Westminster and Central Eye Hospitals and the Ophthalmic Institute (University of London)	55	Behcet's disease, posterior ring abscess of metastatic origin in (Corrigendum)	256
Neuro-paralytic Keratitis	243	Binocular vision	321
On appointment books and appointments	575	Blepharoplasty, contributions to total	108
On bindings	187	"Blue haloes" in atebrin workers	40
On eye shades	442	Bone, glaucoma secondary to fibrocystic disease of the	731
On forewords	245	British Orthoptic Journal	127
On observation	113	Burns of the eye, further experience with amniotic membrane grafts in caustic	409
On second opinions	576	Cataract cases, post-operative security in (Correspondence, 771)	569
On the decay of proof-reading	186	familial, with extensive pedigree chart	385
Patron Saints of the Eyes	442	Chamber, restoration of the, after intra-capsular extraction	572
Spare copies of the earlier volumes of the Transactions of the Ophthalmological Society of the United Kingdom	307	Choroid, a case of metastatic osteosarcoma in the	713
Their name liveth for evermore	443	Ciliary arteries, posterior, the variations in the system of the trunks of	738
The milking-side eye	709	spasm, a survey of esophoria and	285
The surgical team	114		
Through a glass darkly	643		
Tobacco amblyopia and the budget	503		

	PAGE
Coal mines, lighting in ...	254
Coloboma, bilateral, of the optic disc, a case of ...	689
Colour vision in the consulting room (Correspondence, 445, 512, 645, 710) ...	275
Conjunctivitis due to exposure to dimethyl-sulfate (Correspondence, 509, 510) ...	373
welder's ...	774
Contact lens Society ...	56, 384
lenses ...	565
lenses in excelsis ...	304
Copyright material (Memorandum) ...	63
Cornea, respiration of the, in albino rats, some researches on the ...	100
the reticulo-endothelial system of the ...	421
Correspondence ... 123, 191, 249, 313, 383, 509, 646, 710, 771	
"Dam" method, ophthalmic calculations by the ...	424
Dark Adaptometer, an improved ...	235
De Oculis, by Benevenutus Grassus of Jerusalem (Corrigenda, 320) ...	123
Detachment of the retina, technical improvements in a diathermy operation for ...	436
Dimethyl-sulfate, conjunctivitis due to exposure to ...	373
Di-ophthalmology, an aspect of. Peripheral and central disturbances of the visual fields ...	721
Diplopia, the treatment of traumatic ...	581
Egypt, Ophthalmological Society of ...	63
Esophoria and ciliary spasm, a survey of ...	289
Estelle Doheny Eye Foundation. Eye Laboratory at Los Angeles ...	647
Evans blue, the effect of nitrogen mustard on the permeability of the blood-aqueous humour barrier to ...	717
Excerpta medica (Holland) ...	446
Eye-bank for sight restoration (New York City) ...	712
Eye diseases, visual diagnosis of, by means of infra-red radiation ...	419
distribution of penicillin in the, after subconjunctival injection ...	517

	PAGE
Eye, fundus of the, circulatory studies of the ...	238
further experience with amniotic membrane grafts in caustic burns of the ...	409
in a third nerve palsy, a note on the position of the ...	427
mepacrin and the ...	191
removal of the wrong (Correspondence, 250, 316) ...	8
shades (Annotation) ...	245
the effect of aluminium and its alloys on the. A report presented to the Vision Committee of the Medical Research Council ...	449
the regeneration of wounds of external membrane of, in the light of new pathologico-anatomical results (Corrigendum, 516) ...	336
Eyelid, a case of an adenoma arising in a sweat gland of the upper ...	697
Eyes, patron saints of the (Annotation) ...	375
Faculty of Ophthalmologists ...	644
Familial cataract with extensive pedigree chart ...	385
Foreign body, the intra-ocular. A series of 72 cases in the B.L.A. ...	12
Fundus of the eye, circulatory studies of the ...	238
Future of Ophthalmology ...	119
policy (British Journal of Ophthalmology) ...	1
Glaucoma, concussion ...	116
secondary to fibrocystic disease of the bone ...	731
the flap sclerotomy in the treatment of ...	72
the production of a filtering cicatrix in ...	65
Grönblad-Strandberg syndrome ...	228
German measles, pigmentary abnormality in children congenitally deaf, following maternal ...	119
Heterophoria in air crew, the cure of ...	116
Honours ... 62, 127, 319, 516	
Hordeolosis, autohaemo-therapy in ...	766
Human embryo and foetus, trochlear nerve in ...	257

INDEX—SUBJECTS.

	PAGE		PAGE
Hunterian lecture	...	Lids, congenital vertical shortness	...
Hypopyon formation, local penicillin therapy of, with special reference to the use of subconjunctival injection	528	of the, combined with tetraastichiasis, a case of	219
Illuminating Engineering Society	254, 447, 515	Light, white and coloured, the comparative visual acuity and ease of reading in	129
Indians, astigmatism in (Correspondence)	313	Lighting in coal mines	254
Infra-red radiation, visual diagnosis of eye diseases by means of	419	"Lightning streaks," subjective	46
Intra-ocular foreign body. A series of 72 cases in the B.L.A.	12	Lister Morton ophthalmoscope, the use of unit cells	317
pressure, the influence of, on the rate of drainage of aqueous humour. Stabilisation of intra-ocular pressure or of aqueous flow	160	Macular dystrophy, a case of the Laurence - Moon - Biedl syndrome showing atypical retinitis pigmentosa associated with	211
Intra-vitreous use of penicillin and other drugs, experimental observations on the	193	Meningeal reactions in sympathetic ophthalmitis	366
Intra-capsular cataract extraction, its most serious complication	119	Mepacrin and the eye (Correspondence)	191
Iris atrophy, a case of bilateral genuine	176	Mr. Surphlete, an item of ophthalmological history	3
prolapse, an unusual case of symmetrical, bilateral, non-traumatic	700	Moorfields, Westminster and Central Eye Hospitals...	243
Irish Ophthalmological Society	254	Myopia and pseudo-myopia; (Correspondence 124, 249, 383.) Corrigendum 64	198
Keratitis, neuro-paralytic (Annotatation)	575	Marfanil, intra-vitreous use of	198
rosacea, allergy to endogenous hormones as a cause of	145	National Society for the Prevention of Blindness	320, 514, 580, 647, 648
Lacrimal stricturotomy, a survey of the results of...	51	Prize subject: Glaucoma	128
Lamina cribrosa and its nature	551	New Zealand, The Ophthalmological Society of, (B.M.A.)	515
Lamps in coal mines	255	Night vision testing and night vision training in aerial warfare	116
Laurence-Moon-Biedl syndrome showing atypical retinitis pigmentosa associated with macular dystrophy, a case of	211	Nitrogen Mustard, the effect of, on the permeability of the blood-aqueous humour barrier to Evans blue	717
ns, induction of an experimental tumour of the	676	Notes	62, 127, 254, 319, 384, 446, 514, 580, 646, 712, 772
the power or "powers" of a	570	Oculomotor paralysis, position and movements of the eye in. (Correspondence)	511
ses, contact	565	Ophthalmic calculations by the "Dam" method	424
ie Dana Gold Medal award (1947)	304	Institute (University of London)	243
	773	literature (an abstracting service)	2, 772
		Ophthalmitis, on meningeal reactions in sympathetic	366

INDEX.

Original Communications and Clinical, Pathological and Bacteriological Memoranda.

	PAGE
Bakker, A.—Some researches on the respiration of the cornea in albino rats	100
——— The action of sulphanilamide on rabbits' lenses <i>in vitro</i> ...	216
——— Xanthelasmata and autonomic nervous system. A new syndrome ...	686
Bárány, Ernst, H.—The influence of intra-ocular pressure on the rate of drainage of aqueous humour. Stabilisation of intra-ocular pressure or of aqueous flow ...	160
Beattie, P. H.—A consideration of Aniridia, with a pedigree ...	649
Blum, John—See Katzin, Herbert M.	
Boxill, W. M. de C.—The flap sclerotomy in the treatment of glaucoma ...	72
Briggs, Allan H.—Contact lenses in excelsis ...	304
Bromberg, Y. M.—See Zondek, B.	
Campbell, Dorothy—Binocular vision ...	321
Clifton, F. and Gordon, W. H.—A case of an adenoma arising in a sweat gland of the upper eyelid ...	697
Cooper, E. R. A.—The trochlear nerve in the human embryo and foetus...	257
Corcelle, L.—On meningeal reactions in sympathetic ophthalmitis ...	366
Cory, J. W. E.—Glaucoma secondary to fibrocystic disease of the bone ...	731
Cruise, Sir Richard—The production of a filtering cicatrix in glaucoma ...	65
Czukrás, Ida—A case of bilateral genuine iris-atrophy ...	176
——— Contributions to total blepharoplasty ...	108
Davson, Hugh and Quilliam, J. P.—The effects of nitrogen mustard on the permeability of the blood-aqueous humour barrier to Evans blue. An instance of the influence of a lesion in one eye on the susceptibility of the other ...	717
Duguid, J. P., Ginsberg, M., Fraser, I. C., Macaskill, J., Michaelson, I. C., and Robson, J. M.—Experimental observations on the intra-vitreous use of penicillin and other drugs ...	193
Forgacs, J.—See Weinstein, P.	
Fraser, I. C.—See Duguid, J. P.	
Ginsberg, M.—See Duguid, J. P.	
Godtfredsen, Erik—Ophthalmic-neurological symptoms in connection with malignant nasopharyngeal tumours ...	78
Gordon, W. H.—See Clifton, F.	
Gördüren, Süreyya—A Grönblad-Strandberg syndrome ...	228
Handousa Bey—Some observations on the symptomatology and diagnosis of cases of proptosis ...	155
Haridas, Gopal—Wernicke-Tay-Sachs disease in a Chinese infant ...	428

INDEX—ORIGINAL COMMUNICATIONS, ETC.

	PAGE
iv.	
Haythorne, Joan—See Sorsby, Arnold	
Heffernan—See Wolff, Eugene	
Hughes, H. L.—A case of bilateral coloboma of the optic disc	689
Irvine, G.—A survey of esophoria and ciliary spasm	289
James, R. R.—Mr. Surphlete, an item of ophthalmological history	3
Johnstone, I. Lloyd—The development of medical studies in Britain: Ophthalmology	179
(Familial cataract with extensive pedigree chart)	385
(Corrigendum 580)	
Katzin, Herbert M. and Blum, John—Transplantation of vitreous. A preliminary report	760
Kettesby, A.—Restoration of the chamber after intra-capsular extraction...	572
Technical improvements in the diathermy operation for detachment of the retina	436
Koch, Walter—An improvised dark-adaptometer	235
Krwawicz, Tadeusz—The reticulo-endothelial system of the cornea	421
Landau, J.—A case of congenital vertical shortness of the lids combined with tetrastrichiasis	219
See Zondek, B.	
Lees, V. T.—A case of metastatic osteosarcoma in the choroid	713
Levkoieva, E. Th.—The regeneration of wounds of external membrane of the eye in the light of new pathologico-anatomical results	336
(Corrigenda 516)	
Lloyd, Ivor—A survey of the results of lacrimal stricturotomy	51
Lyons, F. Maxwell—Contracted socket. The splint method of post-operative control	703
Macaskill, J.—See Duguid, J. P.	
Magnus, J. A.—A case of pseudo-glaucoma	692
Mann, Ida—"Blue Haloes" in atebirin workers	40
Contact lenses	565
Induction of an experimental tumour of the lens	676
Martin, L. C. and Pearse, R. W. B.—The comparative visual acuity and ease of reading in white and coloured light (Corrigenda 256)	129
Michaelson, I. C.—See Duguid, J. P.	
Moore, R. Foster—Subjective "lightning streaks"	46
Nairac, M. L.—An unusual case of symmetrical, bilateral, non-traumatic iris prolapse	700
Neely, J. C.—The treatment of traumatic diplopia	581
Neubert, Frank R.—Colour vision in the consulting room	275
Parry, T. G. Wynne—Post-operative security in cataract cases	569
Pascal, Joseph I.—Ophthalmic calculations by the "Dam" method	424
The power or "powers" of a lens	570
Pearse, R. W. B.—See Martin, L. C.	
Peleška, M.—See Vaško, A.	
Quilliam, J. P.—See Davson, Hugh	
Reed, Howard—See Sorsby, Arnold	
Robson, J. M.—See Duguid, J. P.	
Roper-Hall, M. J.—Research in Zurich	223
Rugg-Gunn, A.—A case of recurrent aphthous uveitis with associated ulcer.	306
vulvae acutum (Lipschütz)	

INDEX—BOOK NOTICES.

v.

PAGE

Savin, L. H.—The effect of aluminium and its alloys on the eye: A report presented to the Vision Committee of the Medical Research Council ...	449
Somerville-Large, L. B.—Panophthalmitis in a premature infant treated by streptomycin ...	362
Sorsby, Arnold and Ungar, J.—Distribution of penicillin in the eye after subconjunctival injection ...	517
— Haythorne, Joan and Reed, Howard—Further experience with amniotic membrane grafts in caustic burns of the eye ...	409
— and Reed, Howard—Local penicillin therapy of hypopyon formation. With special reference to the use of subconjunctival injection ...	528
Stallard, H. B.—The intra-ocular foreign body. A series of 72 cases in the B.L.A. ...	12
Stern, H. J.—Conjunctivitis due to exposure to dimethyl-sulfate ...	373
— Autohaemo-therapy in hordeolosis ...	766
Stutterheim, N. A.—Peripheral and central disturbances of the visual fields. An aspect of di-opthalmology ...	721
Sudakevitch, T.—The variations in the system of the trunks of the posterior ciliary arteries ...	738
Taylor, Charles—A case of the Laurence-Moon-Biedl syndrome showing atypical retinitis pigmentosa associated with macular dystrophy ...	211
Traquair, H. M.—Removal of the wrong eye ...	8
Ungar, J.—See Sorsby, Arnold	
Vasko, A. and Peleska, M.—Visual diagnosis of eye diseases by means of infra-red radiation ...	419
Weinstein, P., and Forgács, J.—Circulatory studies of the fundus of the eye	238
Wilczek, Marian—The lamina cribrosa and its nature...	551
Wolff, Eugene and Heffernan—A note on the position of the eye in a third nerve palsy ...	427
Zondek, B., Landau, J. and Bromberg, Y. M.—Allergy to endogenous hormones as a cause of keratitis rosacea ...	145

Book Notices.

An Introduction to the prescribing and fitting of Contact Lenses (Frank Dickinson and K. G. Clifford Hall) ...	1946	246
A Treatise on Gonioscopy (Manuel U. Troncoso) ...	Philadelphia, 1947	579
Cirurgia Ocular (H. Arruga) ...	Barcelona: Buenos Aires, 1946	190
Clinical methods of Neuro-ophthalmologic examination (Kestenbaum) ...	New York, 1946	508
Corneal Transplantation (B. M. Filatof) ...	Odessa, 1945	188
Die Erkrankungen des Augenhintergrundes (Dr. Adalbert Fuchs) ...	Vienna, 1943	313
Diseases of Children's Eyes (James Hamilton Doggart) ...	London, 1947	578
Diseases of the Retina (Herman Elwyn) ...	London, 1946	770

INDEX—NAMES.

Eye manifestations of internal diseases (I. S. Tassman)	London, 1946	PAGE 577
Eye Surgery (H. B. Stallard)	Bristol & London, 1946	121
Instrumental Optics (G. A. Boutry)	Paris, 1946	60
Klinische und Erbbiologische Untersuchungen ueber die Heredoataxien (T. Sjögren)	Copenhagen, 1943	311
Les yeux et la vision des vertebres (Rochon Duvigneaud)	Paris, 1943	312
Physiologie Oculaire Clinique (A. Magitot)	Paris, 1946	58
Principles of the Contact Lens (H. Treissman and E. A. Plaipe)		247
Report on defective colour vision in industry, by a committee of the colour group of the Physical Society		247
Sensory Mechanisms of the Retina. With an appendix on Electro-retino- graphy (Ragnar Granit)	Oxford, 1947	376
The Science of Seeing (Ida Mann and Antoinette Pirie)	1946	248
The Surgery of repair injury and burns (D. N. Matthews)	Oxford	310
The 1946 Year Book of the Eye, Ear, Nose and Throat	Chicago	444
Transactions of the American Ophthalmological Society Vol. XLIII	1945	60
Transactions of the Ophthalmological Society of the United Kingdom Vol. LXV	London, 1946	60

NAMES.

Ascher, K. W.	PAGE 251
Bakker, A.	
Bárány, Ernst H.	100, 216, 686
Beattie, P. H.	160
Berens, C.	320, 384, 649
Blum, John	514
Boase, A. J.	760
Boxill, W. M. de C.	772
Brecher	72
Briggs, Allan H.	775
Bromberg, Y. M.	304, 317
	145
Campbell, Dorothy	
Clifton, F.	321
Cooper, E. R. A.	697
Corcelle, L.	257
Cory, J. W. E.	366
Crawford, R. A. D.	731
Cridland, Nigel	315
	124, 445
Cross, A. G.	
Cruise, Sir Richard	PAGE 447
Czukurász, Ida	65
	108, 176
Dansey-Browning, G. C.	514
Davson, Hugh	717
Doggart, J. H.	446
Duguid, J. P.	193
Duke-Elder, Sir Stewart	62, 506, 516
Emsley, H. H.	316
Foote, Franklin M.	
Forgács, J.	320
Fraser, I. C.	238
	193
Ginsberg, M.	
Godtfredsen, Erik	193
Gordon, W. H.	78
	697

	PAGE
Ophthalmological Society,	
American	515
Irish	254
of Australia	115, 712
of Egypt	63
of New Zealand (B.M.A.) ...	515
of the United Kingdom.	
(Annual Congress)	62, 307
(Transactions of)	307, 516
Ophthalmology in South Africa	
(Notes)	384
post-graduate course in ...	63
the development of medical	
studies in Britain:	179
Ophthalmoneurological symp-	
tom in connection with	
malignant nasopharyn-	
geal tumours	78
Optic disc, bilateral coloboma of	
the, a case of	689
Orthoptic Journal, British ...	127
Orthoptic treatment, the rôle of	116
Osteosarcoma in the choroid, a	
case of metastatic	713
Oxford Ophthalmological	
Congress... ..	254, 448
Palsy, third nerve. A note on	
the position of the eye in	
(Correspondence 511)	427
Pan-American Congress of	
Ophthalmology	320, 448
Panophthalmitis in a premature	
infant treated by strep-	
tomyacin	362
Pathological specimens, found	
(Correspondence)	317
Penicillin and other drugs, ex-	
perimental observations	
on the intra-vitreous use	
of	193
in the eye after subconjuncti-	
val injection, distribu-	
tion of	517
therapy (local), of hypopyon	
formation with special	
reference to the use of	
subconjunctival injection	528
Pigmentary abnormality in chil-	
dren congenitally deaf	
following maternal Ger-	
man measles	119
Post-graduate course in Ophthal-	
mology	63
Proptosis, some observations on	
the symptomatology and	
diagnosis of cases of	155
Pseudo-glaucoma, a case of ...	692

	PAGE
Reading in white and coloured	
light, the comparative	
visual acuity and ease of ...	129
Removal of the wrong eye ...	8
(Correspondence)	250, 316
Reticulo-endothelial system of	
the cornea	421
Retina, angeoid streaks of the	119
detachment of the, technical	
improvements in the	
diathermy operation for ...	436
Retinal detachment, inherited ...	118
Retinitis pigmentosa, atypical,	
associated with macular	
dystrophy. A case of the	
Laurence - Moon - Biedl	
syndrome showing	211
Retrobulbar neuritis and de-	
ficiency disease	117
in prisoners of war. A survey	
of	117
Royal Society of Medicine (Sec-	
tion of Ophthalmology)	773
Sociedad de Oftalmologia del	
Litoral	515
Socket, contracted. The splint	
method of post-operative	
control	703
Sodium sulphacetamide, intra-	
vitreous use of	197
South Africa, Ophthalmology in	384
Streptomycin, panophthalmitis	
in a premature infant	
treated by	362
Stricturetomy, a survey of the	
results of lacrimal	51
Sulphanilamide, the action of,	
on rabbits' lenses in	
vitro	216
Synoptophore, the "normal"	
in the (Correspondence)	
... ..	123, 315, 445
Tetrastichiasis, a case of con-	
genital vertical short-	
ness of the lids combined	
with	219
Third nerve palsy, a note on the	
position of the eye in a	
(Correspondence, 511)	427
Thomas Masters, spectacle-	
maker, abstract of the	
Will of, 1625 (Notes)	775
Tobacco amblyopia and the	
budget (Annotation)	503
Trachoma, International organ-	
ization against	774
Trochlear nerve in the human	
embryo and foetus	257
Tumour of the lens, induction of	
an experimental	676

	PAGE		PAGE
Tumours, ophthalmic-neurological symptoms in connection with malignant nasopharyngeal	78	Visual fields, peripheral and central disturbances of the	721
Uveitis recurrent aphthous, a case with associated ulcer vulvae acutum (Lipschütz)	396	Vitreous, transplantation of	760
University of Glasgow, Department of Ophthalmology	646	Waren Tay-Sachs disease in a Chinese infant	428
V.335, intra-vitreous use of	198	Welder's conjunctivitis	774
Vision, binocular	321	Western Ophthalmic Hospital	128
colour, in the consulting room	275	White Oak Hospital, Swanley, Kent, 1944-46	320
night, testing and training in aerial warfare	116	Wounds of external membrane of the eye, the regeneration of, in the light of new pathologico-anatomical results (Corrigendum 516)	336
Visual acuity and ease of reading in white and coloured light, the comparative defects in army recruits	513	Xanthelasmata and autonomic nervous system. A new syndrome	686
		Zurich, research in	223

